New Orleans Medical
and
Surgical Journal

Vol. 98 SEPTEMBER, 1945 No. 3

RUPTURED UTERI: AN ANALYTICAL REPORT OF SIXTY-FOUR CASES MANAGED AT THE CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS WITH A REVIEW OF THE LITERATURE*

WOODWARD DAVIS BEACHAM, M. D.†
NEW ORLEANS
AND
GEORGE ANDREW VARINO, M.D.
MONROE

Since Guillemeau[1] (1550-1613) first described rupture of the uterus, many accounts of this distressingly serious obstetric accident have found their way into print. Doubtless this condition claimed the lives of many women long before it was recognized as a clinical entity, and we dare say that numerous have been the times that the cause of death has been recorded as "childbirth," "childbed fever," "postpartal hemorrhage," and the like, whereas it should have been *rupture of the uterus.*

Our interest in this subject was initiated when we saw a case as students, and we became markedly interested during the first year of our residency in obstetrics and gynecology, during which time nine such cases were managed in the Charity Hospital of Louisiana at New Orleans. We have perused the available literature on this grave obstetric complication and have studied the records of all cases of ruptured uteri treated in Charity Hospital since 1912. When this study was undertaken we received the encouragement of the chiefs of the obstetrical services of Tulane University, the Louisiana State University, and the Independent Unit. Again, we express our appreciation to them and to the members of their staffs for permitting us to report the cases which have been treated on their respective services.

*Presented in part before the joint meeting of the Orleans Parish Medical Society and the New Orleans Gynecological and Obstetrical Society June 1, 1939.

†From the Departments of Obstetrics and Gynecology of Tulane University, School of Medicine, and Charity Hospital of Louisiana at New Orleans.

INCIDENCE

Obviously, statistics regarding the incidence of uterine rupture are difficult to compile; nevertheless, we do know that the frequency is conspicuously higher in localities where pelvic deformities predominate. One must also take into account an undoubtedly large number of fatal cases of rupture which pass unrecognized, with the patient succumbing due to shock, peritonitis, or the like. Furthermore, hospital figures serve only as an index to the general frequency of the accident, because complicated and mismanaged labors gravitate chiefly to institutional practice. Sheldon[2] states, "Fourteen of twenty-six cases of uterine rupture occurring in the Boston Lying-In Hospital from January, 1918, to January, 1935, were emergency cases and cannot be charged against the hospital clinic." Forty-five of the sixty-four cases in the New Orleans Charity Hospital series were classified as emergencies at the time of admittance.

Individual reports as to incidence vary from one in 250 labors (Tauffer[3]) to one in 6,124 labors (Ramsbotham[4]). In 1875, Bandi[5] reported a frequency of 1:1200; and five years later Harris[6] gave as his ratio 1:4000. Referring to cesareanized cases in

Incidence of Uterine Rupture as Reported by Various Authors

Institution Author	No. Rupt.	No. Deliveries	Ratio of Incidence	Institution Author	No. Rupt.	No. Deliveries	Ratio of Incidence
Moscow Maternity Clinic Moscow, Russia (1877-1901) *Ivanoff*[13]	124	118,581	1:956	Rotunda Hospital** Dublin, Ireland	34	57,412	1:1689
Kasr El-Ainy Hospital Kitchener's Memorial and Coptic Hospitals Cairo, Egypt (1903-1931) *N. P. Mahfouz*[14]	110		1:3500*	Los Angeles County Hospital California (1923-April, 1934) *L. G. McNeile R. D. McBurney*[20]	30	17,350	1:578
New York Lying-In Hospital (1909-Sept. 1920) *Asa B. Davis*[15]	106	86,648	1:817	Boston Lying-In Hospital (1918-1934) *C. F. Sheldon*[21]	26	47,554	1:1829
New York Lying-In Hospital (1890-1909) *Ralph Waldo Lobenstine*[16]	78	60,977	1:781	Maternity Hospital Rivadavia (& Inst. of Maternity) Buenos Aires, Argentina (1920-1935) *F. Bazan F. A. U. Imaz*[22]	21	27,500	1:1309
Maternity Hospital Bucharest, Rumania *Cristeanu & Draghiesco*[17]	77	23,016	1:300				
Charity Hospital of Louisiana New Orleans, Louisiana (Jan. 1913-Jan. 1941)	64	57,144	1:892	Woman's Hospital New York *R. A. Hurd*[23]	9	17,500	1:1944
Philippine Is. Gen. Hospital (1910-1933) *B. Roxas, A. Baens, J. R. Katigbak*[18]	56	34,447	1:636	Royal Maternity Charity of London (1827-1856) *Ramsbotham*[4]	8	48,996	1:6124
Koenigsberg Clinic Germany (1910-1929) *H. Sachs*[19]	36	20,800	1:577	Toronto General Hospital (1906-1916) *W. A. Scott*[24]	6	3,973	1:662

*Estimated by Mahfouz as the general incidence in Egypt.

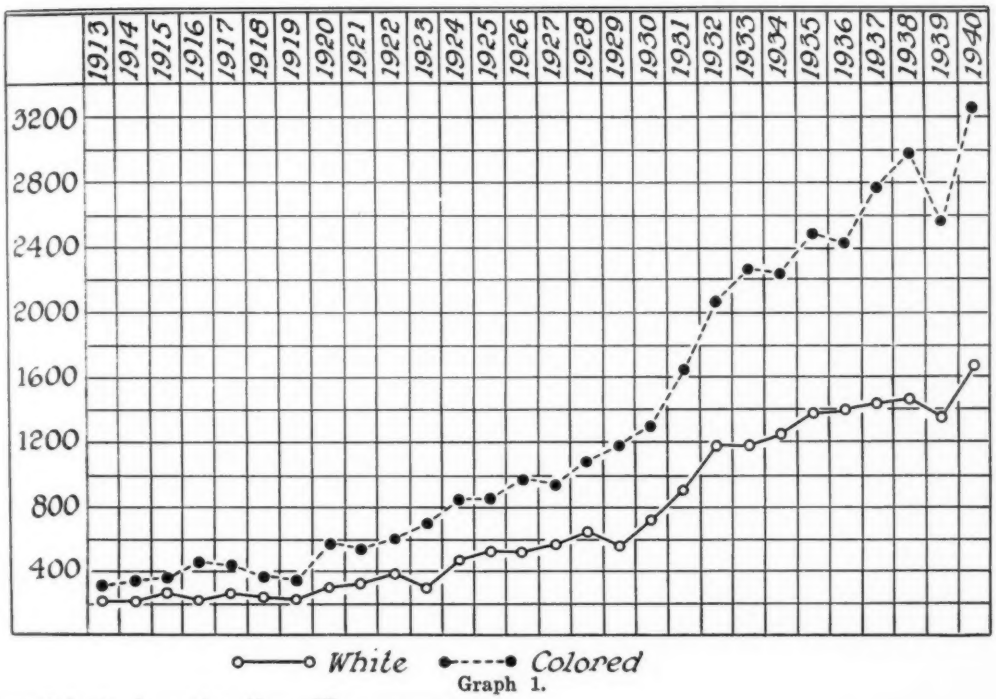
**Cited by P. B. Bland and L. Goldstein in Davis' Gynecology & Obstetrics. Vol. I, Chapter XIX, p. 4, 1938.

Table 1.

1886, Krükenberg[7] declared that after the old operation 50 per cent of all cases resulted in rupture of the uterus during subsequent pregnancies. He reported 20 cases of rupture through the scar, with a mortality of 50 per cent. Leopold[8] (1889) gave an incidence of 1:6100. Freund[9], in 1890, studied the statistics of 17 investigators, arriving at an average of 1:2114. Koblanch[10] (1895) reported 1:462 and Winckel[11] in his 1905 *Handbuch* wrote that the frequency was one in 666 deliveries. In 1914, Rongy[12] concluded that 3 per cent of all cesareanized women are destined to have a ruptured scar with a mortality of 50 per cent. Table 1 shows the ratio of incidence as reported by Ivanoff,[13] Mahfouz,[14] A. B. Davis,[15] Lobenstine,[16] Cristeanu and Draghiesco,[17] Roxas and Katigbak,[18] Sachs,[19] McNeile and McBurney,[20] Sheldon,[21] Bazan and Imaz,[22] Hurd,[23] and Scott.[24]

MECHANISM OF RUPTURE

Due credit must be given to Guillemeau,[1] Baudelocque,[25] Schröder,[26] V. Winckel,[27] Michaelis,[28] and Braune[29] for the foundation of our knowledge of this subject. However, the modern history of rupture of the uterus is based upon the work of Bandl,[30] who first pointed out the difference in function of the upper and lower uterine segments and discussed the clinical significance of the contraction ring in his writings on the "Lower Uterine Segment" in 1875 and 1876. In order to comprehend correctly rupture of the uterus during labor, one must bear in mind the anatomic features of the uterus, and especially of the development of the lower segment. The latter has been the subject of numerous contributions to the literature since the time of Bandl,[30] and today it is generally agreed that it is formed from the isthmus rather than from the corpus or cervix uteri. Munro Kerr[31] states that Stieve's "papers on the subject explain very clearly the features of this specialized area and how early in pregnancy (sixth to eighth week) its walls fold outwards and its cavity merges into the general cavity of the body proper of the uterus." The mechanism of rupture is explained on the basis of excessive retraction of the upper segment with corresponding thinning and attenuation of the lower seg-



Graph 1.

ment in tonic contraction. The upper segment is then stimulated to even more violent activity, and finally thinning has reached such a degree that even the slightest extra strain will result in rupture of the lower uterine segment. This may occur spontaneously or be brought about by outside interference in the form of manual intervention, instrumentation, or the administration of oxytocics.

ETIOLOGY

Gravidity: Etiologic factors leading to rupture of the uterus during pregnancy include anything which may weaken the uterine wall. One of these is multiparity. DeLee[33] stated that the occurrence is about eight times more frequent in multiparae than in primiparae, being "due to structural changes, the weakness of the muscular fibers, the scars and inflammations, the residues from previous labors." In this series there were only seven primigravidae, as shown by table 2. Trask[32] found 31 primigravidae in a collected series of 417 cases of ruptured uteri. Of his 78 cases, Lobenstine[16] stated that 23, including 18 "incomplete ruptures," were in primiparae. In the subsequent New York Lying-In series of 106 cases, Asa Davis[15] reported only eight primiparae. Sheldon[21] found only two primiparae in the Boston Lying-In Hospital series of 26 cases. Mahfouz[14] calculated a ratio of 15 multiparae to one primipara in 110 cases. McNeile and McBurney[20] list only one primigravida in 31 uterine ruptures at the Los Angeles County Hospital.

Parity, Color, and Maternal Deaths

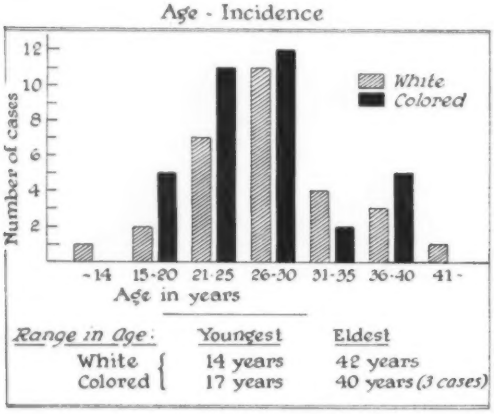
Para	*0*	*I*	*II*	*III*	*IV*	*V*	*VI*	*VII*	*VIII*	*IX*	*X*	*XII*	*?*	*Total*
White	2	5	4	2	1	2	1	2	1	0	1	0	8	29
Deaths	2	3	2	1	0	0	0	1	0	0	1	0	5	15
Colored	5	4	5	2	5	3	2	1	2	2	0	1	3	35
Deaths	4	1	5	0	3	2	2	1	1	2	0	1	2	24

Mortality: 60.93% W-51.77% C: 68.57%

Table 2.

Age: Trask[32] found the largest number of uterine ruptures at the age of 30. He cites one woman who was 47 years old. DeLee[33] listed the years from 30 to 40 as being

those of greatest incidence. McNeile and McBurney[20] state in their article that "there was no predominating age group." Their youngest patient was 20 years and their eldest 41 years of age. Graph 2 shows a



Graph 2.

predominance in the age group from 26 to 30. The youngest case was 14 and the eldest 42 years.

PREDISPOSING FACTORS INCIDENT TO THE UTERUS, FETUS, AND PELVIS

1. *Causes Due to the Uterus*

a. *Uterine Cicatrices*: Injuries of the uterine musculature resulting in scar formation is one of the major predisposing conditions to rupture of the uterus. In the New Orleans Charity Hospital series it will be noted that 17 of the cases had been previously cesareanized. The results of the studies on uterine wound healing by Mason and Williams[34] are well known. They proved that a firmly united scar is even stronger than the uterine muscle, confirming the clinical observations of Schauta,[35] who stated in 1906 that with the modern closure of the wound rupture is more likely to occur outside of the scar. As early as 1904, Van Leuven[36] collected 32 cesareanized cases who subsequently delivered spontaneously without any untoward effects. Since then many much larger series have appeared in the literature, refuting the old adage, "Once a cesarean always a cesarean." However, in this connection it is well to mention the mere fact that a woman who has delivered by the natural passages following a cesarean section does not necessarily mean that she is immune to rupture during subsequent pregnancy. Case No. 50 was subjected to laparotrachelotomy at Charity Hospital in 1935 for "abruptio placentae." Almost two years later she had a "normal" delivery at the same institution, and in 1938 her uterus ruptured spontaneously at term. Operation proved the rupture to be at the site of the low section. Another case, No. 52, was sectioned at a New Orleans hospital for "twins," a classical operation being done. A year later a laparotrachelotomy was performed after "prolonged hard labor." In 1920, the following year, a premature dead fetus was born. In 1924, she delivered a "normal eight-pound baby after a very hard labor," being attended by a midwife. Three years subsequently another low cesarean section was required, and in 1938 she came to Charity Hospital with an impending rupture of the uterus. Actual rupture in progress was seen upon laparotomizing her, the site being in the fundus where the first section was done. Steinke,[37] Ford,[38] Acosta-Sisson,[39] Fava,[40] and Casagrande[41] each report a case of normal delivery occurring in the interim between cesaerean section and rupture. C. H. Davis,[42] Holland,[43] and Thornhill[44] report one case each of a forceps delivery of a living infant in the interval between cesareanization and rupture. Yates and Rezanke[45] and Lazard[46] discuss two spontaneous deliveries following cesarean operation with rupture occurring during the third pregnancy after the section. Both of their cases were at term. McNeile and McBurney[20] report a case of a patient who was sectioned at term and who subsequently had four spontaneous deliveries and a bag induction of labor for eclampsia. Her uterus ruptured spontaneously at the sixth month of the seventh pregnancy. In the literature there are many cases of repeated rupture of the uterus. Case No. 15, previously reported by Gelpi,[47] had a second rupture within a year after her first rupture, following cesarean section, was sutured. Similar cases have been reported by Engels,[48] and Cornell.[49] In 1848, Trask,[32] an American, collected three cases with a second rupture of the uterus,

and Becker,[50] a German writing in 1912, reported one such case of his own and a collection of 27. Maljawinsky,[51] at the Second Moscow Woman's Clinic, discussed a similar case in 1934. Mikhine,[52] writing in a French journal, in 1902, found records of 13 patients with a second rupture, of whom at least six died. He cited Battlehner as experiencing three consecutive ruptures in one woman, and Rose noted the occurrence of four ruptures in one patient.

Although Krükenberg[7] stated in 1886 that he believed that fully 50 per cent of sectioned uteri ruptured during subsequent pregnancies, Sänger,[53] in an 1895 report, presented a collected series of 500 cases without a single rupture. This marked contrast was attributed to the improved technic of suturing introduced by Sänger in 1882. From 1895 to 1908, 20 cases of ruptured cesarean scars were collected by Brodhead,[54] whose elucidative article appeared in the *American Journal of Obstetrics* (1908). Wyss,[55] writing in 1912, attributed the increase in the number of ruptures not to the growing popularity of cesarean section but to departure from the method of Sänger. In 1916, Palmer Findley[56] tabulated 63 cases, including the 20 presented eight years earlier by Brodhead.[54] He included the case of rupture after two cesarean sections reported by Lobenstine[57] in 1908, as did Brodhead; but he did not mention the three cases of ruptured sectional scars which were reported by Lobenstine[16] in 1909, or the case of C. H. Davis[42] reported in 1913. Writing in the same year as Findley,[56] Bell[58] reported a case stating, "From the literature available in the library of the Wayne County Medical Society and the Medical Library of the University of Michigan, I have been able to find seventy-eight cases recorded, my own case making seventy-nine. This included the sixty-three cases tabulated in the very exhaustive paper on this subject in the *American Journal of Obstetrics*, by our esteemed Fellow, Dr. Palmer Findley." As to whether he included the cases of Lobenstine[16] and C. H. Davis[42] we are at a loss to know, due to the fact that cases were not tabulated and no references were given. In 1933, LaMariana[59] reviewed the literature on rupture of the cesarean scar during pregnancy and labor, finding 96 cases subsequent to Bell's[58] report and prior to 1933.

A consideration of predisposing factors and contributing causes of rupture in sectioned cases is in order. Of these we consider the improper healing of the uterine incision as being the most important. In this connection, let us consider location of the incision, infection in the uterine wound, technic of suture and the material used, and location of the placenta.

Location of the Incision: The transverse fundal incision introduced by Fritsch[60] in 1897 soon became discarded. Writing in 1912, Wyss[55] remarked that the transverse fundal incision had not lessened the danger of rupture. Scheffzek[61] voiced the opinion that the unusual tissue distortion in the fundus during puerperal involution makes firm union of the scar problematical. Couvelaire[62] found 17 fundal cases in his report of 50 ruptured scars. Vogt[63] (1911) reported six fundal scar ruptures, and quoted Kuster as having had no rupture in a group of 100 cesareans, Leopold[8] none in 232, and mentions Sänger's[53] collection of 500 cases without a rupture. He concluded that rupture of the extraperitoneal operation scar is rare.

Study of the literature reveals the striking fact that rupture of low section scars is quite rare. As already mentioned, case No. 50 had a laparotrachelotomy followed by a normal delivery and a year later by rupture of the scar at term. We have been unable to find a similar case in the literature. Alice Maxwell,[64] Wetterwald,[65] and Casagrande[41] report one case each of rupture of a low section scar during the next subsequent pregnancy. Wetterwald,[65] in an article in 1926 on the obstetric future of women after low transperitoneal cesarean section, stated that the literature records only 10 ruptures in the cicatrix in 3,600 cesarean sections by this method (0.28 per cent). He found that 45 out of 100 patients cesareanized by the low section technic became pregnant. In 13 cases the operation

was repeated, and the old scar was almost invisible. In the other cases childbirth occurred by the natural route. After high cesarean section, he believes that 25 per cent will rupture in subsequent deliveries. Writing in 1936, Fournier and Estienne[201] reported a case of rupture of the scar during labor following a cervical cesarean section, and stated that Bonnet, in 1934, was able to collect 37 such cases from the literature.

Infection in the Uterine Wound: Infection undoubtedly accounts for the weakness of an untold number of section scars. In many of the cases reported this fact has been emphasized, especially in those of Wyder,[66] Schutte,[67] Myer,[68] Van Buren,[69] Stevens,[70] Hillis,[71] Rickards,[72] Tottenham,[73] and Brandt.[74] LeDoux,[75] Warren,[76] and others report an instance of complete separation postoperatively of the uterine wound attributed to infection. Prine[77] reports a case of utero-abdominal fistula following section. A febrile postsectional convalescence may be considered indicative of uterine wound infection if all other causes of fever are eliminated in the particular case under observation; however, even though the patient is free of fever after operation we cannot then be absolutely certain that there has been no infection in the uterus. As Findley[56] stated, "there is no positive assurance of obtaining a perfect wound healing whatever the method of suture or whoever the surgeon. The uterine scar is an unknown factor in all cases." Hilliard E. Miller[78] has very aptly written, "The deaths from rupture of the uterus after cesarean section of course carry their own lesson, that when a woman has once been delivered abdominally, her life is in jeopardy in all future pregnancies unless the utmost watchfulness is practiced throughout her pregnancy and labor."

Technic of Suture: The exact technic of Sänger[53] has not been followed for many years, but the essential principles of his method of suture have been generally observed. It is justifiable to assume that marked deviations from his teachings have been responsible for a number of ruptures. These well-known rules include tier suturing, exclusion of the decidua in the sutures, infolding of the serosa, and the tying of sutures snugly without too much pressure. If these conditions are maintained and the wound heals in its entirety by primary intention, one can be assured that the uterine wall is no weaker during subsequent pregnancy than it was prior to the cesarean section.

There has been much controversy regarding the suture material for closure of the uterine incision. Holland[43] noted in a collected series of 97 cases (including five of his own) of ruptured cesarean scar that of the 53 cases in which the material for the deep sutures was mentioned, catgut was employed in 41, and silk in 12. He declared, "It would be false reasoning to conclude from these figures that rupture is more liable to occur after catgut than after silk." According to him, prior to 1910 the use of silk was far more predominant than the use of catgut in Great Britain. Since then the reverse is true. Writing in 1920, he stated that the literature showed that the American and German operators greatly preferred catgut, whereas silk was still the choice of the French. At that time he personally advocated the use of silkworm gut for the deep sutures, stating that oftimes the catgut was too quickly absorbed, especially in infected cases, and that silk and linen favor suppuration and may lead to fistula formation. Since then the quality of catgut has been greatly improved. Following the teaching of E. L. King,[79] we presently favor the employment of No. 1 chromic catgut, using a continuous suture for approximation of the inner third of the myometrium extending down to but not through the endometrium, a second layer of figure-of-eight sutures or a continuous suture in the outer two-thirds of the uterine musculature, and a continuous Lembert suture closing the uterine fascia. In instances of a transverse cervical incision two tiers of sutures suffice. A suture of OO plain catgut is employed for peritonealization utilizing the vesicouterine fold. In infected or potentially infected cases, interrupted su-

Type of Rupture in Cases without Previous Cesarean Section - Group A

	White			Colored		
	Cases	Maternal Deaths	Fetal Deaths	Cases	Maternal Deaths	Fetal Deaths
Spontaneous	7	5	5	10	7	7
Traumatic	8	5	4	18	16	17
Pituitary extract alone	1	0	1	1	0	1
Pituitary extract with attempted forceps	1	0	1	0	0	0
Pituitary extract with version and extraction	0	0	0	3	3	3
Pituitary extract with forceps and attempted craniotomy	0	0	0	1	1	1
Forceps alone	4	3	0	5	5	4
Forceps with craniotomy	0	0	0	2	2	2
Bag alone	0	0	0	1	1	1
Manual dilatation cervix alone	0	0	0	1	1	1
Bag with version and extraction	1	1	1	0	0	0
Version and extraction	1	1	1	2	1	2
Attempted version (Bandl's Ring)	0	0	0	1	1	1
Breech extraction	0	0	0	1	1	1

N.B. Case #40, white, was 24 days postpartal after a "normal" delivery. She was examined under anesthetic because of continued bleeding. Rupture was found; patient was hysterectomized.
Case #8, white, was 41 days postpartal after a forceps delivery. She was diagnosed incomplete rupture.
No history as to type of rupture in cases 11 (W) and 21 (C).

Table 3.

tures are used throughout the procedure. As a result of the enthusiasm of Conrad Collins, many of the gynecologic cases on the Tulane service have been given the benefit of cotton sutures, and we have employed such material in a series of sectioned individuals. Although we are keenly interested in various suture materials, we still believe that proper wound healing depends more upon the method of suturing than upon the material employed, a thought expressed by Olshausen[80] and Bumm[81] over a quarter-century ago.

Placental Site: It is the general consen-

sus that placental implantation at the site of cesarean scars may predispose to uterine rupture as the result of the erosive action of chorionic villi. In the 97 cases discussed by Holland,[43] mention of the placental site was made in 51. Of these it was implanted over the scar in 34. He believes that too much importance has been attached to the location of the placenta. Nevertheless, it must be admitted that if there is any tendency toward weakness of the uterine scar it will be rendered more vulnerable by the presence of the placenta. This, in itself, is a point definitely in favor of the low type section. Marinacci[82] described the cesareanized case of a patient who, in the latter months of her next pregnancy, developed a severe intestinal hemorrhage. Laparotomy disclosed a loop of bowel adherent to the site of the cesarean incision with invasion of the gut wall by the destructive action of the chorionic villi, giving rise to the bleeding, which preceded by several days the slow rupture of the uterus.

Effect of Repeated Cesarean Section: Very very few authors advocate sterilization after the first section. A few recommend same after the second operation, notably Jardine and Opitz[83]; and many obstetricians believe that it should certainly be performed after the third. Obviously, this is a matter which should be left to the decision of the wife and husband after giving them the benefit of all the facts in the

Type of Rupture in Previously Sectioned Cases
Group B

	White			Colored		
	Cases	Maternal Deaths	Fetal Deaths	Cases	Maternal Deaths	Fetal Deaths
Spontaneous	10	4	8	6	1	5
Type of Section						
a Classical	9			5		
b Laparotrachelotomy				1		
c Both Classical and Low	1					
This patient had 1 Classical and 2 Low Sections						
Traumatic						
Pituitary Extract alone	1	0	1	0	0	0

Table 4.

case. There are numerous cases on record of individuals who have been subjected to several cesarean sections, and Asa B. Davis had a patient upon whom seven were performed. In 1934, Phaneuf[84] reported two patients of his, each of whom had six cesarean sections. Paul Titus,[85] in his book on *Management of Obstetric Difficulties*, wrote, "The greatest number that I have done on one patient is five." In *Berkeley's*[86] *Midwifery* we find "Cesarean section can be repeated indefinitely, it has been done nine times on the same woman, and the risk when performed under proper conditions is no greater than at the first operation." Munro Kerr[87] in his *Operative Obstetrics* in reply to Rivett,[88] who stated he had operated upon one patient for the fifth time and "had always hoped to do 10 cesarean sections on the same patient," declared, "I wonder if the patient has the same desire to establish a record!" He continues, "I certainly think that a patient who has been subjected to cesarean section on two to four occasions has done nobly by her family and by the state. No matter how safe the repeat operation (lower segment) may be, on every occasion there is risk. After all, she is of more value to her family alive with three or four children than dead with 10 children to her credit."

In this connection it is interesting to note that Heffernan[89] and Bailey[90] reported one case each of uterine rupture occurring after the third cesarean section. There are numerous instances of rupture after a second operation, including the cases of Kickham,[91] Tait,[92] Mackenzie,[93] Lester,[94] Fournier,[95] Lobenstine,[57] Hanson,[96] McNeile and McBurney,[20] Warren,[76] Fitzgibbon et al.[97] Cases Nos. 52 and 33 in the Charity Hospital series had three and two sections respectively, before rupture occurred.

Rupture of the Uterus After Myomectomy: This occurrence is extremely rare. In this hospital many women have been myomectomized before and during pregnancy, without any known instances of rupture. A few years ago we removed a degenerating fibroid from the uterus of a Negress who was three months' pregnant. Her case was followed closely and she was delivered of twins at term without difficulty. In discussing uterine rupture after myomectomy, Munro Kerr,[96] in 1937, said, "I have never observed it, nor do I know

of any colleague who has witnessed its occurrence." He mentions Tesauro,[99] who recorded a case and reviewed the literature in 1932. He emphasizes the fact that Tesauro's paper deals chiefly with the danger of rupture in subsequent pregnancy, and declared, "I have not come across a recorded case of rupture of the uterus in a pregnancy which continued subsequent to myomectomy." Mahfouz[14] states that he has never seen a case of rupture following myomectomy and declares that he has personally delivered 10 patients who have become pregnant after myomectomy. Fink[100] reports the case of a person who was subjected to a myomectomy during her third pregnancy and who later fell down some stairs with a resultant rupture of the uterus into the bladder causing gross hematuria. M. Goulliod[101] states that Döderlein encountered one rupture following myomectomy. In 1933, Hurd[23] wrote about a case of rupture at the site of an old myomectomy following breech extraction.

R. R. Kahle[102] cites a case of ruptured uterus at term following a hysterotomy which had been done 18 months previously for removal of retained secundines. Rosenfeld[103] reports an instance of uterine rupture at the fourth month which occurred in the fundus at the site where a wedge-shaped resection had been performed four years previously.

Any instrumentation, manipulation, or infection which results in permanent weakness of the uterine wall must be considered as a predisposing factor. Long,[104] in 1914, pointed out that the foundation for uterine rupture often dates back to some remote illness or operation, the significance of which is not appreciated at the time of impending rupture. Baisch,[105] in his collected series of 79 cases of uterine rupture during pregnancy, found that 31 cases occurred during the first five months of pregnancy, and in these the uterine wall was diseased. Scar tissue was usually found in cases occurring after the fifth month. He presented much data to bear out the theory that one of the main factors determining rupture during pregnancy is previous injury of the uterine wall. Riddel[106] agrees with this contention. He stresses the role played by multiparity, previous injury, infection, and chronic metritis in causing rupture of the uterus before the onset of labor. Banks[107] and Kane[108] report a case of spontaneous rupture at six and one-half

Record of Presentation, Position, Cervical Dilatation; and Location of the Fetus and Placenta after Rupture -		*Presentation*	*Position*	*Dilatation of Cervix*	*Fetus and placenta delivered*	*Fetus delivered placenta in uterus*	*Fetus and placenta in uterus*	*Fetal head and placenta in uterus*	*Fetus in abdomen, placenta in uterus*	*Placenta in abdomen, fetus in uterus*	*Fetus and placenta in abdomen*
Spontaneous	Group A-17 W-7, C-10	Vertex-8 Transverse-1	LOA 3 LOT 2 ROP 3	Compl. 8 Incompl 3	1	0	4	3	5	0	3
	Group B-16 W-9, C-7	Vertex-5 Breech-1	LOA 4 ROA 1	Compl. 1 Incompl. 4	4	0	0	6	0	2	8
Traumatic	Group A-28 W-10, C-18	Vertex-18 Breech-1 Transverse-2 Compound-1	LOA 6 LOT 1 ROA 3 ROP 2	Compl. 14 Incompl. 5	16	1	5	0	2	1	2
	Group B-1 W-1	Vertex ?		Incompl. 1	0	0	1	0	0	0	0

Table 5.

months and six months, respectively, which they attribute to the effects of infection following abortion. Lawson and Gordon[109] and Lloyd and Ganner[110] discuss spontaneous rupture in multiparae attributed to chronic metritis. Masson[111] describes a case of impending rupture at the cornual end three years after applications of radium (1392 mg. hrs.).

b. *Malposition of Uterus*: This is a potential predisposing cause. Scott[24] discussed Campbell's case of incarceration of a retroverted uterus with rupture during pregnancy at the sixth month. Three decades ago Brodhead,[54] in this country, and Sinclair,[112] in Europe, championed the principles set forth by Wallace,[113] believing that "complete utero-parietal adhesions render repetition of cesarean section simple and easy." They admitted, however, that rupture of the uterus could and did infrequently follow ventro-fixation. Two of the cases reported by Mahfouz[14] in 1937 "resulted from a previous fixation operation." In his cases the posterior surface of the uterus became thinned out during labor and rupture occurred at the level of the lower segment. Spalding[114] reported a case of rupture subsequent to uterine suspension. Ventro-fixation procedures in the child-bearing period have been relegated to the past, in so far as we are concerned.

c. *Cervical Operations*: It is a well-known fact that cervical strictures or stenosis may follow any surgical procedure upon the cervix, and one cannot overemphasize the absolute necessity of the proper post-operative care extending over a long period of time in these cases. Doubtless many women have been hysterectomized solely because someone was indifferent or negligent about dilating the cervical canal repeatedly during the months that followed operation. We know of no case of rupture of the uterus in this locality due to amputation of the cervix, trachelorrhaphy, conization, or cauterization. However, we have seen cases of conglutinatio orificii externi following conization, and instances of dystocia attributable to a Sturmdorf amputation. It is obvious that all women who have had any cervical operation should be carefully observed during subsequent pregnancies.

As early as 1883, Murphy[115] reviewed all instances of postoperative cicatrical atresia of the cervix as a cause of dystocia. Audebart[116] (1898), Guibot,[117] and LePage,[118] in 1903, reported difficult labors following trachelectomy. Writing in 1926, Eastman[119]

Length of Labor and Duration of Rupture

		41 days	36 days	24 days	11 days	7 days	5 days	4 days	72 hours	59 hours	48 hours	46 hours	36 hours	28 hours	25 hours	24 hours	23 hours	20 hours	17 hours	15 hours	14 hours	13 hours	12 hours	11 hours	10 hours	9 hours	8 hours	7 hours	6 hours	5 hours	4 hours	3 hours	2 hours	1.5 hours	1 hour	45 minutes	30 minutes	15 minutes	No labor	Unknown
Spontaneous																																								
Group A	L						1		1			1			1	1		2	1										1	1									5	2
	R													1									1		1		1		1	1	1	1	2	1					4
Group B	L								1									1		1			1		1				1	1	1	1	2		2			4	
	R										1									3												3	2		2			2	2
Traumatic																																							
Group A	L					2			1	1	1		1	2		1	1	1	1	1	2		1			1		1												9
	R	1	1	1	1	1		1	1										1		1		2						2			1	2	1	4	3			3
Group B	L										1																												
	R																																		1				

L = Length of labor R = Duration of rupture

Table 6.

discussed a patient who was subjected to a Sturmdorf amputation during the first three weeks of pregnancy, and whose uterus ruptured subsequently, after she had been in labor less than 12 hours. Meagher[120] tells us of a patient who had two normal deliveries after amputation and uterine rupture during the third delivery, forceps being used.

d. *Congenital Anomalies of the Uterus*: In many uteri that rupture spontaneously during pregnancy there are gross congenital abnormalities. Kane[108] stated that infantilism was present in practically every case of rupture occurring in primigravidae. Baisch[105] described the uteri as being "paper thin" in these cases. According to Thorne,[121] however, Ley's primigravide delivered a normal infant 19 months after having the rent in her uterus sutured, precluding infantilism. Persons with infantile uteri are usually sterile, and infantilism very seldom operates as a factor in causing uterine rupture. It is more apt to cause abortion.

Mahfouz[14] reports two cases of faulty development. One occurred in the rudimentary horn of a bicornate uterus, and the other was a case of uterus bicornis with persistence of the urogenital sinus.

Rupture of bicornuate uteri has been reported a number of times. Kaminester,[122] McNeile and McBurney,[20] Gordon,[123] Truesdale,[124] Tovey,[125] and Pintor,[126] each report one such case. Cornual pregnancy rupture occurs much more frequently, three of the Charity Hospital series falling into that category.

e. *Prolonged Labor*: As a general rule, the longer a patient is in labor the thinner the lower uterine segment becomes and consequently the more susceptible to rupture, either spontaneously or as the result of manipulative interference. Table 7 shows that 21 cases were in labor 20 hours or more, two of them being in labor for seven days and one for five days. Of the previously sectioned group (b), three were permitted to stay in labor 15 hours, and one labored for 48 hours. The labor need not necessarily be "dry" to be dangerous.

Location of Rupture

	Group A		Group B	
	Sponton.	Traum	Sponton	Traum.
Cervix into Lower Segment and Corpus			1	
Lower Segment (39)				
Anterior Wall	1		1	
Posterior Wall			1	
Anterior and Posterior Walls	1			
Lateral Wall	2		3	
Wall not stated	8		16	
Lower Segment and Bladder			2	
Low Section Scar				1
Lower Segment extending into Fundus	1	1	1	
Fundus (21)				
Scar of previous rupture			1	
Operative Scar (section)			15	1
Fundus (no operative scar)	1		2	
Pregnant Horn	3			

All ruptures were definitely complete except in 2 cases, which may have been incomplete.

Table 7.

Hilda Shufflebotham[127] and others have reported instances of spontaneous rupture of the uterus in non-sectioned cases with a completely dilated cervix and with intact membranes.

f. *Invasion of Uterus by Hydatid Mole*: Krellenstine[128] could find only four cases of spontaneous rupture of the uterus due to hydatid mole in the literature up to 1924. He reported one such case. In 1935, McClure[129] was able to find a total of seven cases and he reported the eighth. In 1938, Conway[130] described his case of destruction of the entire fundus uteri by the massive rupture caused by invasion of the villi.

g. *Uterine Neoplasms*: It is interesting to note that not one case in the Charity Hospital series had a rupture due to uterine neoplasm. Fibroids are very common at this institution and innumerable Negresses with myomata palpable through the abdominal wall have delivered without difficulty. A number of cases have been sectioned here due to fibroids in the pelvis. P. H. Williams[131] speaks of a woman who was cesareanized for fibroid and contracted pelvis. At her next labor she was again sectioned and the uterus found to be ruptured at the site of previous cesarean. Schwartz[132] cites a case of a woman who extruded a submucous fibroid through the vagina, the removal of which caused unrecognized uterine rupture with resultant death. Weber[133] tells us of an instance of

rupture of the uterus following version and extraction, perforation being at the site of a gangrenous fibroid. Wood[134] reports a case of rupture of an abscess of the uterine wall during labor. He states that the abscess originated in a degenerating fibroid. Kosmak[135] reports the occurrence of spontaneous rupture in a fibroid-containing uterus. The presence of the tumor was proved after version and extraction, and the rent was in the lower segment. A case of uterine rupture at the site of an intramural myoma after a bag had been introduced to dilate the cervix was reported by Throsby.[136] Carcinoma of the cervix, undiagnosed during pregnancy, may cause uterine rupture. Smith[137] reports complete lower segment tear attributed to cervical malignancy.

h. *Overdistention*: The theory of overdistention was set forth by Paddock[138] and Couvelaire[62] because of the occurrence of uterine rupture in three cases of polyhydramnios. Schwartz[132] reported a case of polydramnios with rupture, but this patient also received pituitary extract.

i. *Placenta*: DeSa[139] and Isbister[140] each report a case of uterine rupture associated with placenta praevia. Cases Nos. 5 and 22 were so diagnosed. Phaneuf[141] reported two cases of the association of abruptio placenta and ruptured uterus, an occurrence observed in No. 32 of the Charity Hospital series. Siegler[146] described such an instance with partial uterine rupture. It is probable that these conditions have been present much more often than has been recognized.

2. *Causes Due to the Fetus*

a. *Malpositions and Malpresentations*: Dystocia and impossible deliveries *per vias naturales* produced by abnormal presentations and/or positions of the fetus are prominent factors in the production of uterine ruptures. Transverse lie of the fetus, and brow, face, and parietal bone presentations are all essential causative agents. Rupture occurs with relative frequency following versions and extractions in such cases which have been neglected for a considerable time. Of the series now being reported, there were three cases (Nos. 1, 23 and 48) of transverse lie of the fetus with shoulder or hand presentations, and one of compound presentation (No. 43), as shown by table 6.

b. *Plural Gestation; and Overgrowth of Fetus*: No. 37 presented twins with rupture at the seventh month, the site being a classical section scar. Isbister[140] reported a case of twins with polyhydramnios, with rupture occurring at seven and a half months. Laffont et al.[198] reported a case of rupture following twins, and Orthner[199] cites a case of twins which were extruded into the abdomen due to traumatism. According to C. H. Davis,[42] Jewett reported a case in which one of the twins was born spontaneously, while rupture occurred before the delivery of the other. Gerrard[286] had a case in which the first twin was delivered normally and the second became impacted with prolapse of the cord and a hand. Attempted version and extraction in the home was unsuccessful; consequently, she was brought to the hospital where extraction was performed and rupture detected. Overgrowth of the fetus with resultant feto-pelvic disproportion accounts for a considerable number of ruptured uteri. In the management of women who have had several babies, the tendency for the feti to increase in size must be kept in mind. Furthermore, the observation has been made that diabetic individuals are prone to have large babies.

c. *Hydrocephalus and Other Abnormalities of the Fetus*: According to Bland and Goldstein,[142] Simpson first pointed out that hydrocephalus is a relatively frequent cause of rupture. Blevins,[143] Romulo,[144] Murray,[145] Acosta-Sisson,[39] Porter,[147] C. H. Davis,[42] and Laffont[149] each report one case of rupture ascribed to this fetal abnormality. Zweifel[150] reported two cases. Mahfouz[14] had four cases in his series of 110 ruptures. Fred Müller[151] recorded five cases of hydrocephalus in 108 cases of rupture; Ames[148] six in 100; Trask,[32] twelve in 417; and Merz,[152] found 18 in 230 cases. All these were collected series, and there may have been some reduplication. Lobenstine[16] had but one case of hydrocephalus in his series of 78 cases of rupture, and in the Charity

Hospital series, there are only two (cases Nos. 13 and 47). Keith[153] found that 16 uteri had ruptured in the birth of 74 hydrocephalic infants; and Schuchard[154] noted 14 in a group of 73.

Fetal malformations are very infrequently encountered as causing rupture. Tottenham and Pillai[155] report uterine rupture in a case of Siamese twins. Kowles[333] tells of a case of rupture following version and extraction of a monster.

3. *Causes Due to the Pelvis.* The pelvic condition most frequently responsible for uterine rupture is the generally contracted pelvis of moderate degree. Michaelis[28] taught that the generally contracted pelvis (justo minor) is the most commonly encountered. Bandl,[51] on the other hand, believed that the simple flat pelvis was the most frequent. The mixed type (asymmetrically contracted) is less frequently responsible. C. H. Davis,[42] tabulating the collective series of Trask, Ames, Bandl, Merz, Von Winckel, Fritsch, and the series reported by Lobenstine, found 598 cases of "contracted or deformed pelvis" in 1342 cases of ruptured uteri. Mahfouz[14] had 52 out of 110 (47 per cent), a ratio of the incidence of pelvic deformity generally agreed by most of the writers. If the deformity is great, its significance is appreciated early and a hard protracted labor is not permitted. In the cases with slightly subnormal pelves, however, recognition of the true status of affairs may be too late. A history of multiparity may serve to lull the observer into a sense of ungrounded security, as we know in many cases there are uterine cicatrices from previous labors which may give way and the size of the baby may be greater than that of its predecessors. This series included four cases (Nos. 53, 54, 56 and 58) of stated contracted pelvis in the non-sectioned group (A) and no statement regarding the pelvis in eight cases (Nos. 2, 3, 11, 16, 21, 31, 40 and 46) of that group. Of the previously cesareanized cases (B) we find five with abnormal pelves (Nos. 4, 6, 12, 49 and 52) and two not stated (Nos. 33 and 36). No mention is made regarding the type of pelvis in the cases of cornual pregnancy (Nos. 7, 51 and 55).

Neoplasms of the bony passages sufficiently large to cause dystocia are fortunately only very occasionally encountered. Ovarian tumors, on the other hand, leading to obstruction of the passageway of the fetus are infrequently seen although far from rare. Larinsky[156] reported a case in which an ovarian cyst was responsible for uterine rupture. Recently, we have cesareanized two women with ovarian tumors obstructing the pelvis. According to C. H. Davis,[42] Albers-Schonberg[157] reported a case where a kidney prolapsed into the pelvis causing rupture of the uterus.

We mention vaginal stenosis, septa, and bands only for the sake of completeness. According to Stander,[158] "among the rare causes of serious dystocia, vaginal neoplasms are worthy of mention, Güder, in 1893, having collected 60 cases from the literature. The obstruction was due to the presence of cystic structures, fibromata, carcinomata, sarcomata, or hematomata, arising from the vaginal walls or the surrounding tissues."

4. *Conditions Due to the Individual*: Several authors have pointed out that the only explanation of rupture in some cases is individual susceptibility. They speak about the fact that the distensibility of the tissues varies with the individual. One thing is certain and that is that women with pendulous abdominal walls are more apt to have ruptured uteri than are their better developed sisters. Case No. 58 is an excellent example of the pendulous abdomen type. Cases Nos. 49 and 52 were quite obese.

5. *External Causes*: The uterus is particularly vulnerable during pregnancy due to its size, position, and its distended condition. Its mobility, elasticity, and the pressure equalizing effect of the liquor amnii serve to protect it from contusions and pressures; nevertheless, rupture has occurred from all sorts of trauma, including: falls, kicks, sudden muscular exertions, and goring by animals. The episode, cited by Trask[32] of a bison bull ripping the fetus

from an Indian squaw is classical. Incidentally, she is said to have recovered.

Manual or instrumental dilatation of the cervix has undoubtedly claimed its full share of ruptured uteri. Porter,[147] Phaneuf,[159] Stamm,[160] and others discuss one case each of rupture of the uterus in which a manual dilatation was done. Williamson[161] reported two cases, and McNeile and McBurney[20] in their series of thirty cases, of uterine rupture, found four such cases. The last mentioned also report six cases following manual removal of the placenta. Acosta-Sisson,[30] Holland,[43] Riddel,[106] Sherrill,[162] Cherry,[163] and Clapper[164] cite instances in which the placenta was removed manually with rupture of the uterus.

Ruptured uteri caused by forceps, bags, embryotomies, and the like have taken the lives of an untold number. In the Charity Hospital series it is definitely known that attempts were made to deliver 18 of the patients at home. Four of these were actually delivered by forceps with resultant rupture. Forceps were unsuccessfully applied in six known cases, and just what was done in the remaining eight was not cited.

Versions and Extractions: Versions and extractions under unfavorable conditions are fraught with grave danger. Lobenstine[16] stated that internal podalic version was the etiologic factor in five of the 46 cases of complete rupture in his series, and that there were three ruptures due to "both version and transverse presentation." Sheldon[2] states that from January, 1918, to January, 1935, 731 internal podalic versions were performed at the Boston Lying-In Hospital, with resultant rupture in 12, an incidence of one to 61. McNeile and McBurney[20] found seven versions in the Los Angeles County General Hospital series of thirty ruptured uteri. Version and extraction were done in seven of the Charity Hospital cases and was attempted on one case, as presented by table 4.

Oxytocics: As early as 1847, Trask[32] cited 16 cases of rupture due to the imprudent administration of ergot, and he declared, "There can be no doubt that the injudicious exhibition of this drug has been the source of infinite mischief. It is difficult to obtain data upon this subject, for few in whose practice such cases occur would be disposed to report them, and those met with in consultation practice are kept secret from motives of delicacy."

In 1895, Oliver and Schaefer[165] first demonstrated the pressor action of the posterior pituitary, and Dale[166] (1906) noted that pituitary extract caused rhythmic contractions of the nonpregnant uterus. Bell,[167] of Liverpool, in 1909, and Foges and Hocstaetter,[168] of Vienna, in the same year, used it to overcome uterine inertia and check uterine bleeding. These investigators initiated the use of the extract after its stimulating effect upon the uterus of a pregnant animal was proved by Frankl-Hochwart and Froelich.[169] Pituitary extract became widely accepted by persons practicing obstetrics, and no distinct limitations to its use seem to have been recognized, some persons going so far as to say that it was devoid of all danger. However, as early as 1913 a note of warning regarding its indiscriminate usage was sounded. Edgar[170] reported that even small doses given during the first stage of labor had caused alarming complications —including rupture of the uterus. He had previously advocated its employment in cases of uterine inertia even though the cervix was incompletely dilated. In discussing pituitrin in labor, Mundell[180] (1916) declared, "It is generally accepted that it has no place whatever in normal labor and should never be used in such. It may be used, with great caution, however, in the first stage of labor, but it is not advised. Its field is in secondary inertia in the second stage of labor, and then only in selected cases and with discretion." Writing in the same year, McNeile[181] discussed his case and fifteen more collected by him, formulating the following conditions: "1. Complete dilatation and effacement. 2. The membranes must be ruptured. 3. Presentation should be longitudinal. 4. In cephalic presentations there should be no deflection of the head and the drug should only be used in vertex and breech presentations. 5. There should be no disproportion between the pre-

senting part and the pelvis. Before the use of the drug the previous obstetrical history should be carefully considered and special emphasis should be paid to the consideration of any operative deliveries. 6. The presenting part should be engaged."

During the past two and one-third decades many contributions concerning the administration of pituitary extract have appeared in the literature. Among these we find Wertenbaker,[182] Grosvenor,[183] Marcondes,[184] Hall,[185] Phaneuf,[141] Rucker and Haskell,[186] Pouliot and Truchard,[187] Maxwell,[188] Sherrill,[162] Mendenhall,[189] and many others. Paul Titus[190] expressed the present consensus of obstetrical opinion when he stated that "the use of pituitary extract during labor is now looked upon as unjustifiable and dangerous, although it is unfortunately true that it is still employed by some as a means of hastening labor."

In this series, eight patients received injections of pituitary extract. Of these, it is interesting to note that seven occurred prior to 1927, and that the only previously sectioned case to receive the oxytocic was being attended by a midwife in 1916.

PATHOLOGY

Anatomically, ruptures are divided into two classes: *incomplete*, involving the endometrium and myometrium, but not the peritoneum, and *complete*, in which the peritoneum is torn. The characteristic finding in the former, or partial variety, is a subperitoneal hematoma. If the tear is sufficiently large the placenta or fetus may lie just beneath the peritoneum. The amount of bleeding depends chiefly upon the site of the rupture, being massive if the base of the broad ligaments or the site of the placenta are involved. In spontaneous ruptures the tear is usually more or less oblique and often transverse, although it may assume any shape; while in traumatic ruptures it is usually longitudinal. The anterior uterine wall is most often involved, and the posterior wall the least common. Case No. 58 presented rupture in the anterior wall and another in the posterior wall (lower segment). In reported series complete rup-

Treatment and Time Interval After Rupture

Spontaneous Ruptures	*Lived*	*Died*
Hysterectomy after 72 hours		1
16 hours		1
12 hours	1	
10 hours		1
8 hours		1
6 hours		1
5 hours	1	
3 hours	1	1
2 hours		3
1½ hours	1	
less than 1 hour		1
? hours		2
Rupture at operation		2
Gottschalk-Portes operation after 15 hours	1	
Suture of uterine tear after 20 minutes	1	
3 hours		1
15 hours	1	
Treated non-operatively:		
Delayed diagnosis: B. welchii peritonitis 28 hours		1
Recognized but patient remained in shock 2 hours		1
1½ hours		1
Unrecognized 72 hours		1
4 hours		2
Evidently ruptured for several months		1

Traumatic Ruptures	*Lived*	*Died*
Hysterectomy after 24 hours		1
14 hours		1
12 hours	1	1
6 hours	1	1
2 hours	1	
1 hour	1	3
30 minutes		1
with repair of bladder injury after 2 hours	1	
Suture of uterine tear after ? hours		1
4 hours	1	
1 hour		1
24 days		1
Treated non-operatively:		
In shock 72 hours		1
1 hour		4
Unrecognized 41 days		1
36 days		1
7 days		1
6 days		1
4 days		1
18 hours		1

No history regarding type of rupture in two cases. One was hysterectomized and lived. The other died on the operating table.

Table 8.

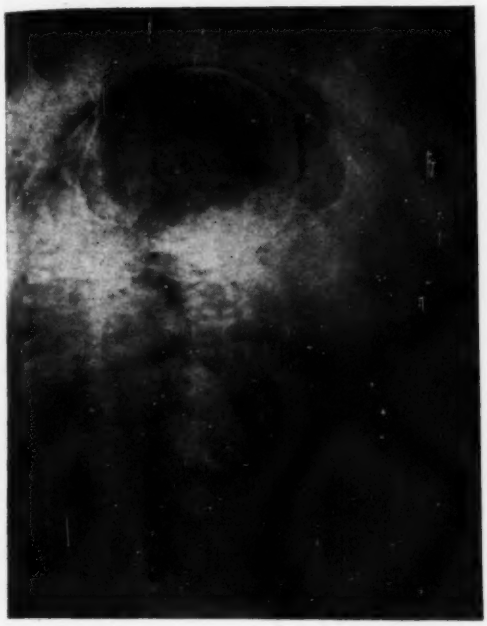
Racial Incidence of Maternal Deaths
Compared with Total Deliveries Per Annum

White			YEAR	Colored		
Number deliveries	Number deaths	Mortality per cent		Number deliveries	Number deaths	Mortality per cent
217	5	2.304	1913	308	8	2.590
202	5	2.475	1914	319	6	1.880
247	4	1.619	1915	370	5	1.351
227	5	2.202	1916	458	6	1.310
242	2	.826	1917	434	5	1.150
227	7	3.083	1918	387	3	.775
211	4	1.895	1919	370	6	1.621
340	10	2.941	1920	587	11	1.872
340	14	4.110	1921	577	14	2.426
396	13	3.282	1922	598	11	1.839
307	4	1.335	1923	700	13	1.857
466	9	1.931	1924	832	14	1.682
510	8	1.568	1925	848	15	1.769
504	6	1.190	1926	974	9	.923
548	13	2.372	1927	964	8	.829
616	11	1.785	1928	1.087	13	1.195
588	10	1.700	1929	1.191	14	1.175
748	14	1.871	1930	1.324	12	.906
907	12	1.323	1931	1.621	17	1.048
1.188	11	.925	1932	2.078	18	.866
1.190	19	1.596	1933	2.263	23	1.016
1.239	9	.726	1934	2.221	22	.990
1.380	8	.579	1935	2.492	20	.802
1.397	12	.858	1936	2.432	16	.657
1.429	7	.489	1937	2.750	28	1.054
1.482	8	.539	1938	2.959	27	.911
1.342	8	.596	1939	2.583	22	.851
1.688	5	.296	1940	3.239	17	.524

Graph 3.

ture is by far more common, and it will be noted that in this series of 64 cases there were 62 definitely known instances. Nevertheless, we believe, as do many others, that the incomplete variety occurs more often than the complete but that it is infrequently recognized. Doubtless many persons presenting a febrile postpartal course, and even some who are fever-free, have minor partial tears which are undiagnosed. Ruptures taking place in cesarean scars are usually complete and may extend into the bladder or cervix or both. Gross hematuria indicates urinary vesical trauma but not necessarily lacerations of the bladder. In the complete class the fetus and placenta may escape into the abdomen or they may plug the uterine rent. (See table 5.) The patient may become exsanguinated or there may be surprisingly little hemorrhage. One of our cases (No. 56), for example, had an annular tear in the lower uterine segment with the fetal head visible through the dilated cervix and with the fetal body free in the abdominal cavity, and yet there was practically no blood in the abdomen and there had been no appreciable bleeding via vaginam. Either of these varieties may occur spontaneously or be due to trauma. In the spontaneous type we have included only those cases that have ruptured without outside interference. By the traumatic type we mean those cases which have ruptured due to intervention (including the administration of oxytocics). Previously cesareanized cases (group B) will be found in both types, as we do not list them as being traumatic solely because they have been sectioned.

Whereas the tears occurring in labor are more commonly located in the lower uterine segment, those making their appearance



Roentgenogram of Case No. 56. Fetal head in the uterus; fetal body in the abdomen; gas bacillus infection.

during pregnancy are, as a rule, confined to the upper part of the uterus. They are usually linear but may assume any configuration. It is rational to believe that they are generally small at first, and that their rapid increase in size is due to uterine contractions. The uterine wall is commonly thinned out or diseased, although there seem to be no definite characteristic microscopic changes applicable to all cases. Hyaline, granular, fatty degeneration, and fibrosis uteri have all been given prominent place by different writers. Simpson,[191] Brindeau,[192] De Lee[193] and others reported fatty degeneration in the muscular fibers in some cases. In recent reports, however, the various writers appear very skeptical about same. Dawidoff and Poroschin[194] found the elastic fibers decreased in number, irregularly thickened, arranged in a zig-zag fashion, and not staining as normal fibers. They attributed this to repeated pregnancies, and cited the work of Martin and Dührssen[195], who described a diminution of the fibers in senile uteri. According to Kerr[196], no subsequent investigations have confirmed the theory of Dawidoff and Poroschin. He declares, "In none of the specimens of my series of 29 cases, submitted to examination, did the late Professor Teacher discover any definite degeneration which could be described as of a particular character. In Mahfouz's[14] series of 110 cases, eighteen uteri were subjected to a very careful examination and in none was there "any essential degeneration."

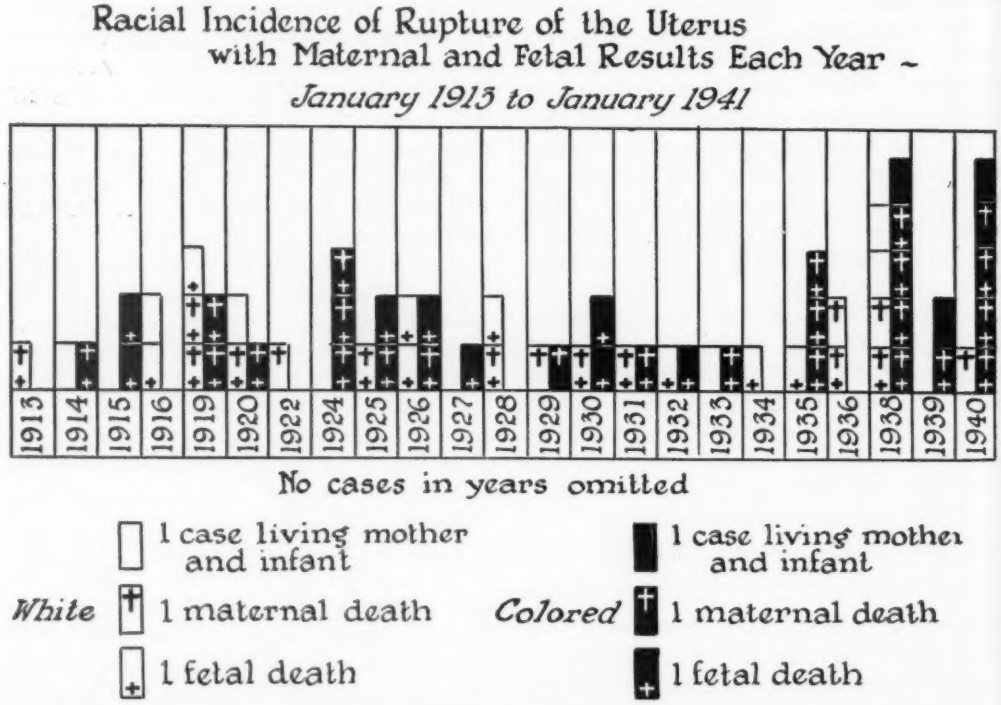
The work of Williams,[197] Williams and Mason,[24] and Losee[200] on uterine wound healing is well-known. In cases of healing by primary intention throughout, there is no cause for concern; however, as has been previously pointed out we can never be absolutely sure that such has occurred in a given case.

SIGNS AND SYMPTOMS: DIAGNOSIS

Impending Rupture: As DeLee[193] has written, "If the accoucher is on the alert, he will almost always be able to detect a threatening rupture of the uterus, and diagnose early a thinning of the lower uterine segment. In contracted pelves and all cases of mechanical obstruction, and where he knows that the uterine wall is weakened, the attendant will be especially watchful." The history in these cases is particularly important, and one should be very careful to inquire into the type of previous pregnancies, kind and duration of labors with management of the third stage, and the presence of any pelvic infection or abnormality. Impending rupture may occur without signs or symptoms, especially in those cases which have been previously cesareanized and who are not in labor. However, the classical picture is usually present, and a diagnosis can be made by thorough observation. Typically, there is no progress in spite of strong uterine contractions, which may be exceptionally painful. Abdominal tenderness, particularly over the lower segment, is the rule. Tenderness over a section scar is of particular significance. Uterine contractions may come one behind the other, and the patient usually shows evidences of restlessness or exhaustion, with an accelerated pulse. The effect

on the blood pressure is variable. The fetal heart tones may become abnormal, but not necessarily so. Bandl's ring may or may not be present.

Actual Rupture: The signs and symptoms of actual rupture range in severity from very mild disturbances to profound shock and death. Before the onset of labor the symptoms may be comparatively mild, and in rupture arising in pregnancy after cesarean section they may also be insignificant. This is the exception, rather than the rule. In spontaneous complete rupture occurring in labor the patient usually suffers sudden, sharp, tearing pain. Not infrequently the patient feels as if something has burst within her. The uterine contractions generally cease if the fetus has been extruded into the abdominal cavity and the patient's "pains" are no longer of the character associated with those of labor, but become more abdominal in type. The backache present during labor may cease. The amount of hemorrhage, with its trend of signs, (falling blood pressure, rapid pulse, "air hunger", blanching of the mucous membranes, decreased temperature, "cold sweats", and even the facies of shock) depends very largely upon the efficiency of tamponade afforded by the fetus, and even to a greater extent upon the location and extent of the rent. Low segment tears not involving the base of the broad ligaments cause less bleeding than those in the fundus. Blood collecting beneath the diaphragm gives rise to shoulder pain, while hemorrhage into the broad ligament may result in pain in the leg. If the fetus is viable at the time of expulsion into the abdomen, the patient may be cognizant of abnormal fetal movements. Abdominal palpation reveals superficiality of fetal parts, and the abdomen may assume very peculiar configurations. The abdomen often becomes rigid, but in case No. 50 marked softness was noted. Nausea and vomiting may occur as premonitory manifestations. There is usually little or no bleeding per vaginam, and vaginal examination discloses a regression of the presenting part. In case

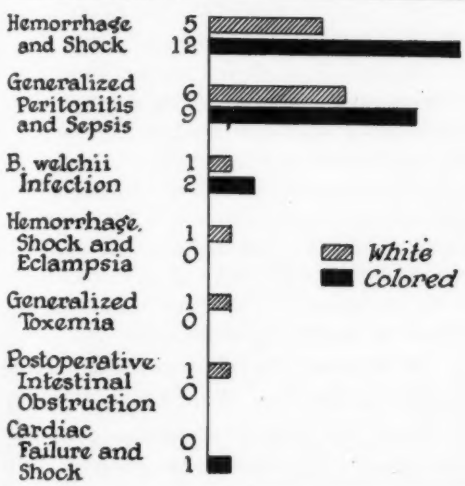


Graph 4.

No. 56 the fetal head was wedged in the pelvis and it was visible through the cervix, although the body was free in the abdominal cavity. In the incomplete variety, emphysematous crackling may be elicited, according to various authors. After delivery, tears may be palpable. Intestine may prolapse into the uterus or vagina in the complete type. Unrecognized ruptures may lead to peritonitis and sepsis, but as we have pointed out small incomplete tears may occur unnoticed.

Any evidence of shock (mild though it may be) during or immediately following labor should call for a prompt consideration of the possibility of uterine rupture. This is especially true if it follows any operative

Causes of Maternal Death



Graph 5.

intervention, the administration of any oxytocic, or if the history contains any facts pointing to weakness of the uterine wall due to previous operations, infections, etc. The cervix should be inspected after every vaginal delivery, and the lower uterine segment should be examined after every operative delivery, if difficulty has been encountered.

PROGNOSIS

The prognosis depends upon the site and extent of the laceration, the associated degree of hemorrhage and shock, the length of labor and duration of rupture, the presence of infection, and the type of treatment. The condition of the mother prior to the

Condition of Feti on Admission to Hospital

Condition	*Number*
Dead	31
Poor	1
Good	20
Non-viable	3
?	4
Delivered alive prior to admission	5
Fetal Mortality	*73.43%*

Table 9.

time of rupture is also a very important factor.

Everyone agrees that uterine rupture is one of the gravest accidents encountered in obstetric practice. In the Charity Hospital series of 64 cases the maternal mortality was 60.93 per cent while the fetal mortality was 73.43 per cent. Tables 10 and 11 set

Condition of Mothers on Admission to Hospital

Condition	*Number*
In shock	21
In early shock	5
Cardiac failure	1
Toxemia (marked)	2
Poor	5
Fair	14
Good	16
Maternal Mortality	*60.93 %*

Table 10

forth the condition of the patients and their feti on admission, accounting for such a high mortality. Roxas, Baens, and Katig-

bak[18] reported a series of 56 cases at the Philippine General Hospital with a maternal mortality of 85 per cent and a fetal mortality of 100 per cent. McNeile and McBurney[20] state that of the 30 cases at the Los Angeles County General Hospital (January 1, 1923 to April 1, 1934) twenty (66.6 per cent) mothers and 19 (63.3 per cent) feti expired. At the Boston Lying-In Hospital (1918-1934) the mortality rate was 42.3 per cent for the mothers and 82 per cent for the babies, according to Sheldon[2], who reported 26 cases (eight of which were incomplete ruptures). In Lobenstine's[16] 46 cases of complete rupture (New York Lying-In Hospital, 1890-1909) there was a maternal mortality of 73.9 per cent and a fetal mortality of 83 per cent. The figures for 29 cases of incomplete rupture were 27.5 per cent and 52 per cent, respectively. Asa B. Davis[15], reporting the ruptured uteri cases at the same institution from 1909 to September, 1926, found

Patients Delivered Prior to Admission and Those Subjected to Attempts at Delivery in the Home

	White	Colored
Delivered by Physicians with Forceps	4	1
Attempts at Delivery by:		
Physicians (with forceps)	2	4
Midwives and Physicians		3
Midwives	1	3
Total	7	11

Table 11.

53.4 per cent maternal and 85.2 per cent fetal mortality in 88 cases of the complete variety. Of 18 incomplete cases there were 55.5 per cent maternal deaths and 61.1 per cent fetal deaths. In his analysis of twelve cases at St. Luke's Hospital in Cleveland and 33 cases occurring during a five-year period in 41 of the 47 member hospitals of The Hospital Obstetric Society of Ohio, Burkons[3,17] calculated a maternal mortality rate of 37 per cent and a fetal fatality incidence of 69 per cent. Rupture after cesarean section carries a better prognosis for the reason that diagnosis is made quicker and treatment is instituted more readily. In the Charity Hospital group there were fifteen previously sectioned patients, four of whom succumbed (26.6 per cent).

TREATMENT

Prophylactic treatment cannot be over emphasized, as it must be admitted that most uterine ruptures are avoidable. As Asa B. Davis[15] said, "The time to treat rup-

Record of Transfusions

Number of transfusions	Lived	Died
0	13	23
1	9	8
2 or more	3	8
	25	39

Table 12.

ture of the uterus is before it occurs. In the last analysis it means better, more conscientious and systematic care during pregnancy, labor and the postpartum state." The injudicious use of oxytocics and any operative intervention of a questionable nature, are to be condemned. The patient who has had a cesarean section or who gives a history indicative of previous serious obstetrical abnormalities should be informed as to the possibilities in her case. It is not necessary to frighten these women in order to convince them that antenatal care is of the utmost importance; on the other hand, they should be assured that their cooperation is a prerequisite for the

Final Diagnosis Determination

	White	Colored
Number Autopsies	6	10
Postmortem Sections and Examinations	2	1
Expired on Operating Table	1	4
Operated upon, No Autopsy	3	5
Diagnosis Proved by Examination (No operation or autopsy)	2	4

Table 13

correct management of their cases. It would be preferable that many such pa-

tients be hospitalized before they go into labor or reach term.

Active treatment depends upon the individual case: In general, hysterectomy should be performed immediately after rupture. The shock and blood loss associated with the accident must be combatted by the administration of blood transfusions. If compatible blood or plasma is not obtainable at the time the emergency arises, infusion of 10 or 20 per cent dextrose in water or gum acacia is of great value. Morphine, oxygen, adrenal cortical hormone, and other anti-shock measures are important, but the value of blood transfusions and plasma infusions is unquestionable. Undoubtedly some of the fatal cases in this series might have survived had they received blood. This is not said in a spirit of criticism as compatible donors were not available in many instances. However, in the present day of blood and plasma "banks", no obstetrical case should be permitted to expire because of the lack of blood. Cases which have been mismanaged, including those that have been examined repeatedly, should be given a double prophylactic dose of tetanus antitoxin and gas gangrene serum. In certain cases one may consider suturing the tear, keeping in mind the possibility of infection and the great jeopardy of rupture during subsequent pregnancy.

Indications of impending uterine rupture are indications for immediate laparotomy. Patients with threatening rupture or ruptured uteri at home should be hospitalized and treated without delay.

The postoperative treatment will include: blood transfusions, infusions, vitamin administration, and measures to combat abdominal distention. Oxygen is definitely indicated in some cases. Chemotherapeusis should be instituted if there is active or probable infection.

SUMMARY AND CONCLUSIONS

1. Sixty-four cases of rupture of the pregnant uterus managed at the Charity Hospital of Louisiana at New Orleans from 1912 to 1941 have been analytically reported.

2. A study of those records and a review of all of the available literature confirms the universally accepted opinion that uterine rupture is a distressingly grave obstetric complication.

3. The high maternal mortality (60.93 per cent) and fetal fatality (73.43 per cent) in this and other large hospital series can be explained in part on the basis that complicated and mismanaged labors gravitate to institutional practice. Forty-five of the Charity Hospital cases were designated as emergencies at the time of admission. In many instances the fetus was dead when the mother arrived.

4. Any evidence of shock during or following labor should call for a prompt consideration of the possibility of ruptured uterus. If suc ha patient responds only temporarily to anti-shock measures the probability of active bleeding is marked.

5. Rupture of the uterus after "low" cesarean section is rare. Nevertheless, every cesareanized individual is entitled to especially close supervision during each subsequent pregnancy. One woman in this series who had been subjected to laparotrachelotomy experienced an uncomplicated delivery via vaginam and a year subsequently the section scar ruptured at term.

6. Classical section cicatrices are particularly vulnerable to rupture, providing foundation for the statement "once a classical section—always a section." A woman in this group had undergone one classical and two low operations. Rupture occurred at the site of the classical incision.

7. Uterine rupture subsequent to myomectomy is quite rare. Although the number of fibroid cases at Charity Hospital is strikingly great, there is not a single recorded instance of rupture following myomectomy. Incidentally, practically all of the myomectomized cases have been delivered vaginally.

8. Successful management of rupture of the uterus requires alert evaluation of the status of affairs and immediate therapeutic action. This series and a study of reported cases substantiate the belief that hysterectomy is the procedure of choice. The administration of blood, and in many cases plasma also, plus other anti-shock

measures cannot be overstressed. The availability of blood and plasma "banks" has been the answer to many obstetrical patients' prayers. We contend that every case should be given the benefit of blood typing and matching during pregnancy.

9. As Asa B. Davis declared, "The time to treat rupture of the uterus is before it occurs. In the last analysis it means better, more conscientious and systematic care during pregnancy, labor, and the postpartum state." The injudicious use of oxytocics and instrumental intervention of a questionable nature are to be condemned.

Acknowledgment: We are very grateful to Dr. E. L. King, Chief of the Tulane University Obstetrical Service, for his valuable advice regarding this subject. Furthermore, we wish to thank the Director and the Medical Records Librarian of the Charity Hospital of Louisiana at New Orleans, for making available the records at that institution and for the figures set forth in Graphs I and V.

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THE RUPTURE (INTRAPERITONEAL, INTRA-INTESTINAL AND INTRAVESICAL) OF SUPPURATIVE PELVIC MASSES

WITH AN ANALYSIS OF 53 CASES FROM CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS

HILLIARD EVE MILLER, M.D.*
NEW ORLEANS

Since Simpson's[1] demonstration of the superiority of expectant over radical therapy in pelvic inflammations, most gynecologists have taught and practiced the conservative management of pyosalpinx and tubo-ovarian abscess. They have also believed and taught that this plan of management is perfectly safe because rupture of inflammatory pelvic masses is an unlikely event if not, indeed, a true surgical curiosity.

Though I still believe that the conservative treatment of acute pelvic inflammations is the preferred treatment for such conditions, my complacency in regard to the possible rupture of inflammatory pelvic masses has been rudely shattered. In a review[2] of the gynecologic deaths at Charity Hospital of Louisiana at New Orleans over a recent six year period I found that of 38 deaths due to tubo-ovarian or pelvic abscess, 14 were demonstrated by necropsy to be due to rupture of these masses, and on clinical grounds the same cause of death could reasonably be presumed in seven other cases in which necropsy was not permitted.

Disregarding for the purposes of this discussion the cases not proved by postmortem examination (though naturally, for practical purposes, they cannot be disregarded), it was both astonishing and disconcerting to find that on a large gynecologic service over a representative period of time the rupture of a pelvic mass was the proved cause of death in more than a third of all fatalities from pelvic inflammation. It was so astonishing, in fact, that I declined to accept the statistics until I had personally studied the postmortem records, all of which were so carefully detailed that their accuracy could not be doubted.

The lesson thus unexpectedly learned led me to examine all the fatal cases of pelvic inflammation in which autopsy was performed at the New Orleans Charity Hospital for the 17 year period ending December 31, 1943, which includes the six year period of my original investigation. For comparative purposes I also examined all the cases of pelvic inflammation treated by surgical measures during the six year period mentioned. I have thus identified (table 1) 44 ruptures of inflammatory masses into the peritoneal cavity, six into the intestinal tract, and three into the bladder, as well as 97 cases in which suppurative pelvic masses were accidentally ruptured into the peritoneal cavity by manipulations at operation. For convenience of

*Dr. Miller died April 20, 1945.

TABLE 1

Distribution of Pathology and Site of Rupture in Spontaneous and Accidental Rupture of Suppurative Pelvic Masses

Location of Rupture	Number of Cases	Pyosalpinx	Ovarian Abscess	Tubo-Overian Abscess	Pelvic Abscess	Pyometrium
Intraperitoneal	44	2	3*	37		2
Intra-Intestnial	6			6*		
Intravesical	3			2	1	
At Operation	97	2	5	85	5	

*In 1 case combined with rupture of a pelvic abscess.

discussion each of these groups of cases will be considered separately.

I have no exact statistical background for these cases because of certain practical difficulties connected with securing such data, including a change in the record system of the Hospital midway in the period studied. As a matter of fact, knowledge as to the exact proportion of cases of pelvic inflammation in which these ruptures occurred would not be particularly useful. It is known that they form only a small fraction of the pelvic inflammatory masses treated, and a still smaller fraction of the thousands of cases of pelvic inflammation treated, at the New Orleans Charity Hospital during the period in question. The important consideration obviously is not the proportion of patients who suffer rupture of pelvic inflammatory masses but rather a realization of the fact that rupture is a definitely possible complication of suppurative pelvic disease, and of the additional fact that it is attended with a tragically high mortality.

INTRAPERITONEAL RUPTURE OF PELVIC MASSES REVIEW OF LITERATURE

The paucity of the literature on the subject and the fact that most instances of rupture of pelvic masses are reported as single cases explain why the majority of gynecologists regard this accident as I myself once regarded it, that is, as a surgical curiosity. Up to 1912, according to Brickner[3], 91 cases had been reported, the first in 1868 by Lawson Tait, who recorded additional single cases in 1875 and in 1883. Most cases were reported as single instances, though Cotte and Challier reported five and Boldt six personally observed cases. Boldt, incidentally, saw no additional cases in the 23 years which elapsed between his report in 1889 and Brickner's review of the literature in 1912. Sabadini,[4] who reported two personally observed cases in 1932, stated that approximately 150 cases had been recorded prior to that date, and Feeney,[5] who apparently did not review the literature in detail, estimated in 1940 that the recorded cases then numbered about 250.

I am unable to say definitely how many cases have been reported since Sabidini's review of the literature in 1932, since most of the contributions are in the foreign literature, most of which was not available to me. Cases reported in the American and English literature in the interim include two by Frank[6] in 1933; one by Endreny[7] in 1936; two by Jensen[8] in 1936; two by Tessitore and Potekin[9] in 1938, these being from the New Orleans Charity Hospital and included in the series which I am reporting; two by Martz and Foote[10] in 1939; three by Haultain[11] in 1939; and one by Feeney[5] in 1940. The titles make clear that the cases reported in the foreign literature are chiefly single cases, and it seems fair to say that the series herewith reported from the New Orleans Charity Hospital is the largest yet to be put on record.

Undoubtedly the accident is very much more frequent than the literature indicates, just as the number of cases not verified by operation or by autopsy is undoubtedly very much greater than the number of verified cases. Many cases are probably diagnosed as ruptured appendicitis or as peritonitis of various other origins, or are not diagnosed at all, while in some cases the rupture may be overlooked because it is walled off, just as, for the same reason, occasional ruptures of the appendix are overlooked. Finally, the number of recorded cases is probably much larger than the figures I have cited from the literature would indicate; they merely cannot be found when the literature is reviewed because the titles of the papers in which they are described do not suggest the contents of the communications. The cases I have listed as reported by Jensen, Haultain and Frank were casually included in discussions of other phases of pelvic inflammation. The cases mentioned by participants in the discussion of Warner's[12] communication on ruptured pelvic masses in 1921 suggest that most gynecologists have actually encountered the condition one or more times but have not troubled to record it.

What all of this amounts to is that the accident is by no means the surgical curiosity which it is commonly stated to be.

Brickner,[3] when he reported his first case in 1912, wrote that even allowing for unreported cases and for cases protected by localized peritonitis, the condition is sufficiently common to be borne in mind in every case of purulent tubal infection. His second communication[13] on the subject, published in 1913, after he had himself operated on two additional patients and had learned of two others in the practice of other physicians was entitled "Rupture of Pyosalpinx Not a Rare Accident."

ANALYSIS OF CASES

Age and Race: Excluding the two cases of pyometrium which ruptured into the peritoneal cavity and which will be discussed separately, the age range in the 42 inflammatory adnexal masses which ruptured in the same site (table 1) was from 16 to 62 years, and 17 patients were 40 years of age or older. In Brickner's[3] collected series the range in the cases in which the age was stated was from 13 to 59 years, and nine patients were over 40 years of age. A number of other patients in whom the accident was reported were in middle life or older, and the age incidence is worth emphasizing because of the general though incorrect belief that infectious pelvic disease occurs almost exclusively in young women in the early childbearing years.

Race is not mentioned in most of the cases reported in the literature but it may be assumed that the majority of the patients were white. In the Charity Hospital series rupture occurred in 33 negro women, as compared with nine white, which is about the proportion in which pelvic inflammation occurs in the two races.

Etiologic Factors: In two cases in the Charity Hospital series the onset of symptoms occurred immediately after a fall. In another particularly clearcut case symptoms began almost immediately after the patient, while hanging up clothes in the yard, was butted in the abdomen by a goat. In three cases the onset followed so closely upon pelvic examination that a cause and effect relationship seems undoubted; probably a number of other cases would have fallen into this category had the records been more complete. In six cases the patients, all negroes, took purgatives following the onset of pelvic pain. Whether in these instances purgation precipitated rupture, or whether rupture had already occurred and was merely aggravated by the medication, it is not possible to say. There seems no reason, however, why purgation under such circumstances would not have somewhat the same deleterious effect as it has in appendiceal inflammation.

Opinions differ as to the importance of trauma as a factor in the rupture of pelvic masses. Some writers suggest that rupture is unlikely in its absence. The accident has been reported after lifting a child; after climbing stairs; after blows of various sorts over the abdomen; after abortion and labor; after coitus; and after gynecologic procedures such as dilatation and curettage of the uterus and the use of cupping glasses on the cervix. It seems fairly frequent, as in the Charity Hospital series, after bimanual examination; in one of Brickner's[3] cases it occurred after pelvic examination on the operating table, while the patient was being anesthetized for operation. Frank[6] reported a most unusual case in which an adnexal abscess ruptured 36 hours after a third tubal insufflation, each of which had revealed the fimbriated end of the tube to be closed.

Pathology and Bacteriology: Twenty-one of the 42 patients had recent infections lasting, on the basis of the clinical picture, from a few days to four weeks. The other 21 had had recurrent acute attacks, and many were being cooled in preparation for surgery. Although positive statements cannot be made on this point, it seems reasonable to assume that rupture is more likely to occur in fresh cases, when the tubal wall is thin and adhesions are either lacking or are fragile, than in older cases, in which thick adhesions have developed. Rupture seldom occurs at the uterine cornu but may occur at any other point. The opposite tube and ovary are frequently though not always involved in the inflammatory process. Gangrene of the adnexa, which is reported in occasional cases in the litera-

ture, was found in one of the Charity Hospital cases, and thrombosis of the iliac veins was found in two instances. In one case a ruptured ovarian abscess was associated with a ruptured pelvic abscess. When the rupture was on the right side a contiguous inflammation of the appendix was occasionally observed.

Bacteriologic studies are scanty in the literature, and my own series is unfortunately also lacking in this respect. In come cases in which examinations were made cultures were sterile, and such cultures as were positive revealed the organisms, in a few instances, cases in which they were made, and showed pneumococci and *E. coli communis*, respectively, in the other two.

Clinical Picture: Clinically the cases at Charity Hospital fall into two groups, just as do the cases reported in the literature. One group presented all the symptoms and signs of an acute abdominal catastrophe, including violent pain, vomiting, and tenderness and rigidity, usually associated with shock. In the second group the symptoms and signs were chiefly those of acute pelvic inflammation, including pain, nausea and vomiting, menstrual and urinary disturbances, diarrhea, chills and fever, tenderness, rigidity and distention. In the extremely acute cases it was usually possible to determine the exact moment at which rupture occurred. In less acute cases it was usually impossible to determine the time of rupture, nor was it always possible in either group to determine which symptoms and signs were due to the basic pelvic disease and which to the superimposed accident.

Diagnostic Considerations: The literature indicates that the diagnosis of a ruptured pelvic mass is seldom made either before operation or, if operation is not done, before death, and the Charity Hospital series substantiates this observation. The diagnosis was set down as a possibility in only two of the 42 cases, and carried no conviction in them, though ruptured viscus, including perforated peptic ulcer, was considered as a diagnostic possibility in 12 other cases. Phases of the clinical picture, as well as the failure to consider rupture of pelvic masses as a possibility, are evident in the variety of diagnoses considered, which included, in addition to pelvic inflammation, ectopic pregnancy; tubo-ovarian abscess; fibroids, which were actually present in six cases; ovarian cyst with twisted pedicle; septic abortion; septic endometritis; pneumonia; typhoid fever; and acute appendicitis, which was perhaps the most popular diagnosis.

The known presence, followed by the demonstrated disappearance, of a pelvic mass is the most helpful diagnostic sign, but it was present, according to Sabadini,[4] in only five of the cases reported prior to 1932. Three of these cases were recorded by Boldt, whose diagnostic achievement seems without equal. The disappearance of a known mass was noted in two Charity Hospital cases, but the significance of the observation was unfortunately not realized.

Brickner[3, 13] suggested as a useful prodromal symptom the presence of repeated attacks of increasingly severe pain for longer or shorter periods, to be explained by a sudden increase of tension in the abscess sac, "ulcerative advances" in the tubal wall, or "progressive rupture" in the absence of protective adhesions. The violent pain which presumably indicated the actual rupture was preceded in 13 instances in the Charity Hospital series by exacerbation of the chronic pain from which the patients had suffered from varying periods of time, but the observation was usually made too late to be of any diagnostic value.

Management: In spite of the evidence of the Charity Hospital statistics as to the possibility of rupture of pelvic masses, I still believe that we are correct in treating conservatively the usual patient with pelvic inflammation. I think, however, that we shall have to revise our ideas as to how long conservatism is safe, and I know that we shall have to observe our patients, both in and out of hospital, with much greater care if we are to avoid more of these tragic accidents.

Certain cases in the Charity Hospital

series bear out these statements. Thirteen patients were in the Hospital when the accident occurred, all of whom died except one who was operated on immediately after the mass had been ruptured during a bimanual examination; the only other patient in this group who was operated on died four hours after operation and nine hours after the rupture. Another patient who had been sent home to cool preparatory to operation, after seven days in hospital, returned in shock 24 hours later and died within 11 hours. Still another patient applied for admission for abdominal pain, was examined, and, after a tuboovarian abscess was found, was sent home to cool. She suffered her rupture on the way home, the accident apparently having been precipitated by the pelvic examination. She was operated on within two hours but died 14 hours later. Many of these cases seem to support the statement made by a number of writers that the most unsafe place to suffer a surgical catastrophe is in the hospital, under direct medical observation. Others bear out Young's[13] warning, that patients with pelvic masses should not pass from observation without operation unless they and their families are warned of the possible risk of rupture.

When rupture has actually occurred, the patient's life depends on prompt operation, and Brickner's[3, 13] point seems well taken, that recurrent attacks of increasingly acute pain in a patient with known pelvic inflammation should be regarded as in indication for immediate operation, with the object of forestalling rupture. Removal of the affected adnexa is obviously the procedure of choice. Feeney[5] advocates only drainage, for two reasons, that pus does not continue to discharge from a tube, as it does from the appendix, and that an intense inflammatory reaction in lymphadenoid tissue does not occur in the tube, as it does in the appendix. Sabadini[4] advocates disinfection of the peritoneal cavity and the use of a Mickulicz drain. In spite of his report of two cases successfully treated by this method, I question its wisdom.

Twelve of the 42 patients with ruptured pelvic masses at the New Orleans Charity Hospital were operated on. Simple drainage was carried out in eight cases, with six deaths; salpingo-oophorectomy in two cases, with one death; and removal of the adnexa combined with supravaginal hysterectomy in another case, which was fatal. In one of the two remaining cases adhesions were released, the exact pathologic process remaining unrecognized until necropsy, and in the other, in which the rupture was also unrecognized until necropsy, ligation of the vena cava was carried out, in an attempt to control thrombosis of the iliac vein.

Sulfanilamide or one of its derivatives was used locally or parenterally in nine of the 16 cases treated in 1939 and thereafter. Powdered sulfanilamide was instilled into the peritoneal cavity in three surgical cases, with survival in one instance, and parenteral chemotherapy was used in two other surgical cases, in both of which the patients survived. It is highly significant that these three patients are the only ones who survived in the whole group of 52 cases, and equally significant that chemotherapy in the absence of surgery apparently does not alter the course of events in rupture of pelvic masses.

Prognosis: The categoric statement can be made that in the absence of prompt surgery the patient's doom is apparently sealed. Even when operation is done the mortality is very high. Some of the patients in the Charity Hospital series died within three or four hours of the onset of the symptoms and signs which indicated rupture, several died on the operating table, and no patient whose exact time of rupture could be determined lived longer than 48 hours without operation.

The Charity Hospital statistics parallel those in the literature. In the 91 cases collected by Brickner[3] in 1912 the death rate was 100 per cent in the 36 non-surgical cases and almost 33 per cent in the 55 surgical cases, the mortality rising proportionately according to the length of time which elapsed between operation and rupture; there were only seven recoveries in the 27

cases operated on after 48 hours. The surgical mortality in the series collected by Sabadini[4] in 1932 was 45 per cent. Martz and Foote[10] in 1939 stated that the original over-all mortality of 50 to 60 per cent had declined approximately 20 per cent in the preceding 25 years. I can see no reason for such optimism, nor does their own statement support it, that no case is on record of recovery without surgery, and that death is the rule if operation is delayed more than 48 hours. The routine use of chemotherapy, as the Charity Hospital figures suggest, should greatly improve the chances of a patient in whom the diagnosis is made and operation is performed without delay, but diagnostic suspicion, unfortunately, does not seem to have kept pace with advances in therapy.

Rupture of Pyometrium: The two cases of rupture of pyometrial collections into the peritoneal cavity via the tube are of considerable interest. The first patient, a 45 year old negro woman, had an enormous abdominal enlargement which had developed over a five month period. She had lost 50 pounds and presented an extremely poor surgical risk. Exploratory laparotomy revealed an inoperable condition of undetermined origin but assumed to be papillocystadenoma of the ovary. Postmortem examination, however, showed that leiomyomato had undergone red degeneration, resulting in a pyometrial collection which extended into the tube and ultimately ruptured into the paritoneal cavity. The second patient had carcinoma of the cervix, which had been treated by radium, and was in the hospital for terminal care. As in the first case, a pyometrial collection extended to the tube and ultimately ruptured into the peritoneal cavity. Both cases represent a possible though apparently unusual complication of pyometrium, which could be prevented by prompt recognition of the intrauterine suppuration and the prompt institution of drainage.

Accidental Rupture of Pelvic Masses at Operation: Over the six year period ending in 1942 the records of the New Orleans Charity Hospital show that suppurative pelvic masses were accidentally ruptured at operation in 97 cases (table 1). Seventy-eight patients were negro and 19 white. The age range was from 17 to 56 years, and 47 patients were 40 years of age or older. Six died, four of peritonitis, one of shock on the operating table, and one, probably, of embolism. In 30 cases the spill was stated to be massive, but only one of the six deaths occurred in this group. The procedure in the fatal cases included unilateral removal of the adnexa in two, and bilateral salpingo-oophorectomy and hysterectomy in four. Appendectomy was done in one of the latter cases, and on the surface seems a most unwise procedure, in view of the contamination of the peritoneal cavity by the spill of pus. On the other hand, 21 other patients whose appendices were removed under the same circumstances made smooth recoveries.

The absence of serious consequences in most of these cases, as compared with the catastrophic consequences of rupture of pelvic masses in the group of cases previously discussed, is open to several explanations: (1) many, though by no means all, of the patients have been thoroughly cooled before operation, and the pus, on the basis of the findings in similar cases, can be assumed to have been sterile; (2) the general peritoneal cavity was usually well packed off when the rupture occurred; (3) the accident in every case was recognized at once, and the spilled pus was promptly and probably adequately removed by suction and other methods; (4) intraperitoneal chemotherapy was used in most of the recent cases. Three of the six deaths occurred before this practice was general, the fourth patient died on the operating table, and it is perhaps significant that chemotherapy was not employed in either of the two remaining fatal cases. Drainage or its omission seems to have played no special part in the outcome, since it was not used in most of the 97 cases. Whether or not these explanations are correct, it seems clear that the rupture of pelvic masses at operation in no wise approaches the seriousness of the accident when it occurs spontaneously.

INTRA-INTESTINAL RUPTURE OF PELVIC MASSES

Like all surgeons, I have personally observed a number of cases in which a pelvic mass ruptured into the rectum and the patient, as a result, was spared the necessity of surgical drainage. The outcome of rupture into the intestinal tract, however, is not always so good. In six such cases at Charity Hospital there were five deaths. Two patients were white and four negro, and the age range was from 25 to 36 years. All were in poor condition when they were first seen. A brief summary of each case is appended:

1. The only patient who survived was submitted to abdominal drainage for long-standing pelvic suppuration, following which a fecal fistula developed. At the second operation, at which time she weighed 71 pounds, it was found that a left tubo-ovarian abscess had ruptured into the signoid. After a stormy convalescence following bilateral salpingo-oophorectomy, complete hysterectomy, excision of the fistulous tract, and repair of the sigmoid, she eventually recovered completely.

2. The patient was submitted to colostomy for intestinal obstruction as the result of pelvic inflammation. Several months later, when an endeavor was made to clear out the pelvis and close the colostomy, it was found that a tubo-ovarian abscess had ruptured into the ileum. Death occurred shortly after operation, probably because the operation performed was far too extensive for a thoroughly toxic subject.

3. The patient was submitted to colostomy for intestinal obstruction as the result of pelvic inflammation, dying shortly afterward. A tubo-ovarian abscess which had ruptured into the rectum was found at postmortem, and a review of the history seemed to indicate that the accident had occurred about 48 hours before operation.

4. The patient, on the third day after colostomy for intestinal obstruction due to pelvic inflammation, complained of violent abdominal pain, went into shock, and died within a few hours. Postmortem examination revealed a fresh rupture of a left tubo-ovarian abscess into the rectum.

5. The patient died on the operating table and it was not clear when rupture of a tubo-ovarian abscess into the rectum had occurred.

4. The patient died suddenly of thrombosis of the basilar artery, and at postmortem examination communications were found between the rectum and a pelvic and a right tubo-ovarian abscess. In neither this nor the preceding case is it clear how much part the rupture played in the fatal outcome.

There is not a great deal to be said about these cases except to emphasize again that rupture of a suppurative pelvic mass into the intestine is not necessarily a favorable event for the patient and is best prevented by timely and adequate drainage of the mass. It must be granted, however, that this accident is by no means uncommon and is frequently not lethal; it probably occurred in a number of other nonfatal cases at the New Orleans Charity Hospital which were not located by the methods used in this investigation.

INTRAVESICAL RUPTURE OF PELVIC MASSES

Rupture of suppurative pelvic masses into the bladder and other portions of the urinary tract is not a usual event, though in view of the regional anatomy and the frequency of suppurative pelvic processes, it is curious that it does not occur more often. Apparently the vesical wall is singularly resistant to contiguous infection.

The number of recorded cases is small. Falk and Hockman,[14] who collected 21 cases from the literature in 1939, added three personal cases, and Donohue[15] recorded another in 1940. The latter case was a remarkable instance of scarlet fever followed by metastatic infections in various parts of the body, including peritonitis, an ovarian abscess, a mastoid abscess, and finally a pelvic abscess which ruptured into the bladder.

All three cases observed at the New Orleans Charity Hospital (table 1) occurred in white women, whose ages ranged from

23 to 36 years of age. In two instances it was possible to determine from the patients' histories exactly when the rupture had occurred. In both instances excision of the diseased pelvic structures was followed by prompt closure of the fistula, in one instance involving the bladder and in the other the intraligamentous portion of the right ureter, which showed a cystic dilatation almost its entire length. The third patient had had a cauterization of the cervix five weeks after an early abortion and had developed a pelvic abscess which ruptured through the vagina. Chemotherapy, and cul de sac drainage twice, at intervals of 18 months, produced only transient improvement, and the failure was explained by subsequent cystoscopic demonstration of the vesical fistula, the bladder capacity having been reduced to four ounces. The patient declined to consider surgery and left the hospital in such poor condition that death probably occurred shortly afterward.

SUMMARY AND CONCLUSIONS

1. The records of the Charity Hospital of Louisiana at New Orleans show that during a recent 17 year period 44 pelvic masses ruptured spontaneously into the peritoneal cavity, six into the intestinal tract, and three into the urinary tract. During a recent six year period 97 inflammatory masses were accidentally ruptured into the peritoneal cavity in the course of operation.

2. A review of the literature shows that most spontaneous ruptures of pelvic inflammatory masses are reported singly or in small groups, a circumstance which has probably supported the concept that the accident is rare. Actually, while it is uncommon in contrast to the frequency of suppurative pelvic processes, it is apparently an important cause of death in pelvic inflammations.

3. Without operation the mortality in spontaneous rupture is 100 per cent, and unless operation is done very promptly the surgical mortality is also high. The death rate in pelvic masses ruptured accidentally at operation, though higher than the general death rate for pelvic inflammations treated surgically, does not approach the death rate of spontaneous rupture.

4. The justification for such a report as this lies in the statement made by Brickner in 1912, that ". . . it is worth while to record all instances observed in order to determine the clinical features of this disastrous accident and to learn, if possible, how an impending rupture may be recognized and forestalled."

5. The fact that the rupture of suppurative pelvic masses is not uncommon and that the accident is attended with a tragically high rate should not be interpreted as an argument for the radical treatment of pelvic inflammations but rather as a warning that patients in whom conservative therapy is practiced must be kept under constant careful observation.

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INFILTRATION THERAPY OF SUBACROMIAL BURSITIS with CALCIFICATION

MAJOR I. W. KAPLAN
AND
MAJOR B. L. HAWKINS
MEDICAL CORPS, ARMY OF THE UNITED STATES

While it is difficult to determine the exact incidence of subacromial bursitis with calcification, our experience on the orthopedic service of a Station Hospital leads us to believe that the condition is quite common.

Calcium deposits in the soft tissues about joints have in recent years become a major surgical problem, both in civilian and military life and has been variously reported as bursitis calculosa; calcifying bursitis; tendinitis calcificans; peritendinitis calcarea; perarthritis; but most frequently as bursitis with calcification.

By far the most common site of calcified tendinitis is the subdeltoid location. Following in frequency are the trochanteric and the medial tibial collateral ligament near the adductor tubercle, often called Pellegrini Stieda's disease.

Growing experience with the general condition of so-called calcareons tendinitis seems to indicate rather convincingly that it is a real clinical entity, having a typical pathology and symptomatology.

PATHOLOGY

Pathologic studies have been reported of the various areas in which calcification occurs, and the findings have been quite consistent. Practically all seem to indicate that the condition is secondary to a degenerative change in the tendons, or adjacent ligaments or capsular structure. The most conspicuous histologic feature is the degenerative change in the fibrous tissue.

The degeneration may vary in degree, from trivial loss of microscopic detail in the connective tissue, to a true necrosis. In such areas apparently because of deficient blood supply, calcium is deposited in the form of calcium phosphate or of calcium carbonate, or a molecular combination of both. The calcified areas increase in size, coalesce to form larger masses which lie in cavities in or near the tendon.

In 1908, Codman, operating on a subdeltoid bursitis, made the important observation that the deposit was not in the bursa, but beneath its base, and in or adjacent to the tendon of the supraspinatus. Since that time many others have substantiated the intratendinous location of the deposits. The large subdeltoid bursa lies between the deep surface of the deltoid muscle, the acromial arch and the lateral surface of the shoulder joint. The supraspinatus tendon which is fused into the superior part of the capsule of the shoulder joint, forms much of the floor of the bursa. It is therefore evident that the primary pathology is in the supraspinatus tendon, and that the bursa becomes involved only secondarily. The motion of the bursa over the calcified area in the tendon and pressure on abduction against the acromial process cause it to become irritated, swollen, and painful. The deposits have been known to rupture through the floor of the bursa into the bursal sac.

SYMPTOMS

The patient with a subacromial bursitis with calcification may complain of pain in the shoulder, often radiating into the forearm and hand; of subjective numbness of the upper arm; of inability to carry out certain movements, especially placing the hands in the hip pockets; lifting an object; combing the hair; or of pain at night which awakens him and forces him to find a more comfortable position. The course of the illness is variable and may in some cases extend over a period of years; spontaneous recovery may occur, but often not until after several months of pain and disability.

PHYSICAL EXAMINATION

Any motion of the shoulder that produces motion of the supraspinatus tendon causes pain. This is most marked in abduction, and external rotation, and least in internal rotation. There is usually a localized area of tenderness on pressure over the greater tuberosity, just below the acromial process.

With the x-ray it is possible to diagnose the existence of calcified deposits accurately. It will reveal them in the tendon though

they are usually interpreted as lying within a bursa.

We should state that although the x-ray studies reveal no calcification a bursitis might still exist, without calcification. Such a condition can be ruled out by infiltrating the bursa with 1 per cent novocaine, and if all the symptoms subside, the diagnosis is probably a bursitis without calcification.

The condition must be differentiated from: (1) arthritis of the acromioclavicular joint; (2) bicipital tendovaginitis; (3) supraspinatus tendon tears; (4) subcoracoid bursitis, and (5) scaleneus anticus syndrome.

TREATMENT

For the past year, on the orthopedic service of the Station Hospital, all cases of subacromial bursitis with calcification have been treated by infiltration therapy. The technic is simple, and one does not have to be a specialist to carry it out. Neither does it require any special equipment. The tender spot in the shoulder is located and the skin over the area is infiltrated with 1 per cent novocaine. The infiltration is carried down to the cortex of the head of the humerus. With the needle in place its position is checked either by the fluoroscope, or by x-ray plates to make certain the needle is in or near the calcified deposit. This is most important. Failure to do so, and doing the procedure blindly, is the cause of many failures. Though we have done a fairly large number of cases, we still often find the point of the needle some distance from the deposit, when we insert the needle blindly and later check the position by the fluoroscope. After adequate analgesia has been obtained and with the point of the needle in or about the deposit, the point of the needle is withdrawn two or three millimeters, and an attempt is made to aspirate calcareous material. This is usually not successful, and we have come to the conclusion that aspiration and washing out of the bursa is not necessary, as repeated postoperative roentgenograms made at regular intervals will show the gradual disappearance of the deposit within several weeks. With the needle in place, 30 to

Fig. I. Roentgenogram of right shoulder taken on 12-3-43 showing a large dense calcareous deposit.

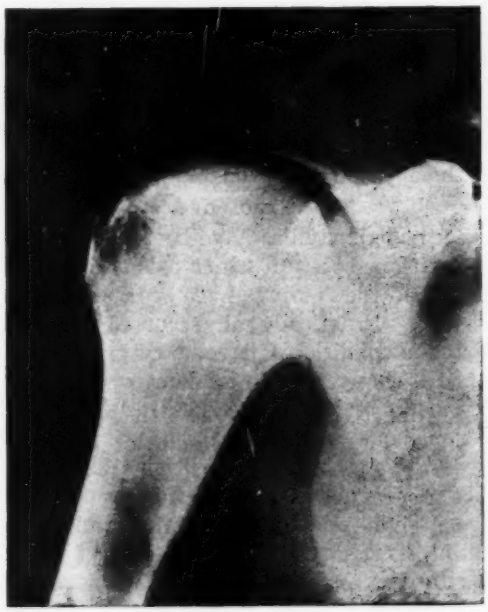

Fig. II. X-ray picture eight weeks after infiltration therapy, showing complete disappearance of the deposit. (Photography by U. S. Army Signal Corps.)

50 cubic centimeters of warm saline solution are slowly infiltrated into and close around the deposit, at the same time numerous punctures are made into the bursa.

The treatment, if applied correctly, usually gives immediate relief, only to be followed by an increase in symptoms for the first 24 hours. We encourage our patients to get under the shower and limber up the shoulder by mild exercise for the first few days. Usually after four days there is complete relief of pain. Serial postoperative roentgenograms made at regular intervals will show disappearance of the deposit, usually within six to eight weeks. Occasionally it is necessary to repeat the procedure if the deposit does not disappear or show decrease in size after four to six weeks, or if symptoms persist. Symptoms disappear long before the deposit is absorbed. However, if the deposit persists, we repeat the procedure, until there is no further x-ray evidence of the deposit being present.

In the past year, on the orthopedic service of the Station Hospital, eighteen cases of subacromial bursitis with calcification were seen. The average age of the patient was thirty-four years. Only four gave a history of trauma. The duration of symptoms varied from ten days to three years. The right shoulder was involved fourteen times, the left four. Two patients had bilateral calcifications. Five patients of the group required more than one treatment, of these four were given two treatments, and one three. Three patients who had two treatments had no decrease in size of the deposit and did not return for further therapy. The patient who had three treatments, showed the deposit decreased in size considerably. Repeated roentgenograms still show a small deposit present.

Of the patients treated, 77 per cent responded favorably, the average time of disappearance of symptoms was six days, of the deposits, seven weeks.

COMMENT

The calcium deposits in these cases of subacromial bursitis, or more accurately, calcified tendinitis, had undoubtedly been present long before the symptoms. One of our patients gave a history of having symptoms only ten days. It is hardly probable that calcifications developed in such a short period. However, that they can develop in a comparatively short time is illustrated by the following case. One of our patients, a prisoner of war, complained of pain in his knees, x-rays were negative, six weeks later the patient was re-rayed, this time there was a calcified deposit in the medial tibial collateral ligament near the abductor tubercle.

The mechanism which precipitates the acute symptoms, as well as the general etiology remain entirely obscure. Infections and trauma are mentioned most frequently as possibilities. The fact that fourteen out of eighteen cases occurred in the right shoulder leads us to believe that the additional trauma of use in right handed individuals might be an important factor.

CONCLUSIONS

1. Subacromial bursitis with calcification most commonly occurs at the right shoulder joint in right handed individuals.
2. The average patient age in our group of cases was 34 years.
3. The trauma of use and infection are mentioned as possible etiologic factors.
4. Infiltration therapy has given excellent results in our series of cases presented.
5. Treatment is accompanied by relief of pain, and by gradual disappearance of the deposit.

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TULAREMIA

ROSCOE L. PULLEN, M. D.†
and
BYRON M. STUART, M. D.†
NEW ORLEANS

The widespread distribution of tularemia, the more general awareness and increasing recognition, the atypical expressions leading to difficulties in diagnosis, the prolonged morbidity and case fatality rate (in our series,[1] 7.55 per cent) and the large number of insect and animal reservoirs of the disease establish it as of more than academic importance. Tularemia should be considered in the diagnostic possibilities of a large variety of acute febrile illnesses, such as influenza, psittacosis, atypical pneumonia, brucellosis, typhoid, tuberculosis, infectious mononucleosis, leukemias, lymphomas, fungous infections and diverse diseases of various types accompanied by septicemia and bacteriemia. Despite the appearances of tularemic buboes and regional lmyphadenopathy, tularemia does not long remain a localized disease but soon develops a general septicemia. Like plague, therefore, tularemia should be regarded as general sepsis in which both bacteriemia and septicemia appear. Consequently the clinical manifestations may be variable indeed and it is the purpose of this discussion to summarize the methods of diagnosis and the treatment of tularemic infections.

CLINICAL MANIFESTATIONS

Incidence. Since the discovery of tularemia as a "plague-like disease of rodents" among California ground squirrels by McCoy[2] in 1911, recognition of the disease has become increasingly frequent so that it is estimated[3] that 2,000 cases annually are reported in the United States at the present time. It is doubtful, however, that this represents an apparent increase of the disease but merely increasing recognition. In the Charity Hospital of Louisiana at New Orleans, 225 cases of tularemia were observed in the period from January 1, 1928, to July 1, 1944. The greatest number of cases occurred in 1940 with the next highest incidence in 1935. For some unexplainable reason there has been a considerable fluctuation of incidence from season to season and from year to year. In recent years, there has been a progressive, significant reduction of the number of cases which may be correlated in part to the reduction of hunting during recent seasons as well as increased knowledge of prophylactic measures among the lay population. We should point out that cases of tularemia are seen in the hospital wards at all times of the year, although the greatest number obviously occur in December, which is at the height of the hunting season.

There are no significant differences in racial susceptibility, negro and white patients being equally affected. Likewise, both sexes and all ages may readily acquire the disease although males by virtue of their occupational pursuits show a slight preponderance. Thus, the youngest patient in our group was a white girl of the age of three years who was bitten by a captive wild rabbit and the eldest patient was a colored male of 73 years.

Contacts. Tularemia occurs most frequently among those occupational groups who handle infected animal flesh, such as hunters and trappers, butchers, cooks and housewives, or those exposed by the nature of geographic location or occupation to the bite of deer flies, ticks or stable flies. Tularemia is a widespread septicemia among a large section of the animal population of this country, hence historical inquiry into the number and type of contacts must consider a wide variety of possibilities. In general it is estimated that 90 per cent of the infections in the United States follow exposure to the tissues or secretions of the cottontail rabbit, jackrabbit and snowshoe hare. The literature, however, records a large group of rodents, gallinaceous birds, and such animals as the dog, cat, coyote, fox, hog and sheep. In addition, ingestion of infected meat and transmission by such insect vectors as the horse fly and deer fly, the dog tick and the wood tick, fleas and

†From the Department of Medicine, Tulane University of Louisiana School of Medicine and Charity Hospital of Louisiana at New Orleans.

lice are responsible for an occasional case of tularemia. The diversity of the animal contacts capable of transmitting the disease to man account for a varied location of the primary lesion and regional lymphadenopathy, a point which we shall emphasize in a moment.

Clinical Picture. As originally proposed by Francis, it is customary and convenient to classify tularemia into four different types, depending upon the predominant expressions of the disease process: (1) ulceroglandular; (2) oculoglandular; (3) glandular; and (4) typhoidal or febrile form of disease. Clinical recognition of the first two forms is not usually difficult if the examiner obtains a definite history of recent animal contact. Recourse to the laboratory is essential for the diagnosis of the latter two types. It should be emphasized, however, that this classification is entirely a clinical one and that the manifestations of tularemia, as with any acute infection characterized by both a septicemia and a bacteriemia, are variable indeed.

The incidence of the various forms may be deduced from our studies of 225 cases as follows: ulceroglandular, 181 cases or 80.4 per cent; oculoglandular, 7 cases or 3.1 per cent; glandular, 23 cases or 10.2 per cent; and typhoidal, 14 cases or 6.2 per cent. The percentage of glandular cases in our group is higher than the average recorded in the literature (3 per cent) which suggests that the primary lesion in many of our cases may have been overlooked either by the physician or the patient before coming to the hospital. As a result, the type frequency of the ulceroglandular variety in our group is lower than the experience of others (87 per cent).

Following an incubation period of two to six days (average for our series, 4.6 days), the onset of tularemia is rapid, acute and characteristically severe with generalized body aches and pains, chills, fever (up to 104° F.), headache, nausea and vomiting, muscular prostration and profuse sweating. Within 48 hours, the primary lesion or buboes appear. As a rule the primary lesion precedes the development of the buboes. The location of the primary lesion varies, depending upon the mode of transmission. In our series of 225 cases of tularemia, a primary lesion developed in 188 cases. In 175 instances it was located on the hands or fingers, and was about equally divided between the two hands. Multiple primary lesions developed in four patients, and in one patient four separate primary lesions were noted. Seven of our patients were instances of ophthalmic tularemia with the development of a severe conjunctivitis. The remainder of the primary lesions were observed in varying locations, such as the forehead, right shoulder, right axilla, right knee, right elbow and left inguinal region. These latter instances were the result of tick bites. There were no instances in our group of oral or pharyngeal involvement, which are infrequent sites in the literature. As just mentioned above, not all patients develop a primary lesion, 37 of our group of 225 cases failing to do so.

Following the papular stage, ulceration of the primary lesion occurs which cannot be distinguished by its clinical appearance alone. A central necrotic core is liberated, the edges of the ulcer are elevated, and the base is ragged and necrotic. Rarely can *Bacterium tularense* be isolated from the lesions by ordinary bacterial smears. Secondary infection occurs frequently. The ulceration is usually painful and quite indolent, the shortest healing time in our group being nine days while the longest was 104 days, averaging about 32 days for the entire series. We may point out that the ophthalmic variety consists of a severe conjunctivitis with redness, congestion, photophobia and lacrimation.

Regional lymphadenopathy or the formation of buboes is part of the clinical picture of tularemia. Usually the process consists of a progressive, painful, tender enlargement of the lymph nodes and in the beginning at least they are usually discrete and firm to touch. One particularly characteristic feature of tularemia is the absence of visible lymphangitis between the primary lesion and the regional buboes, unless severe secondary infection of the primary lesions

has occurred. Extension of the disease beyond the regional lymph nodes may occur and it is not unusual to find generalized lymphadenopathy or bilateral lymphadenopathy when only one ulcer is present. Ttypical lymphadenopathy occurs sufficiently often that tularemia must be considered in the diagnostic possibilities of generalized or other unexplained lymphadenopathy. To illustrate, one of our female patients manifested a nodular type of lymphadenitis which extended from the axillary nodes along the pectoralis muscle to involve the breast. Malignancy was suspected and a biopsy was taken. Another instance of confusion was that of a white male with lymph node enlargement in the left supraclavicular fossa which was thought to be metastatic from a primary malignancy elsewhere. Cervical lymphadenopathy follows the ophthalmic infection with tularemia. In our experience cervical lymphadenopathy in the other varieties is infrequent, occurring in but 14 instances of our entire series. In passing we may state that the spleen is palpable in almost half of the patients with generalized lymphadenopathy as well as those patients with the typhoidal form of the disease. The liver was felt less often but enlargement does occur.

Suppuration of the lymph nodes occurred in 22.2 per cent of our group. Such glands either ruptured spontaneously or were incised after evidences of imminent rupture appeared. Subsidence of the lymphadenopathy in tularemia is very slow, averaging 46.4 days in our group with a range from 17 to 219 days.

As stated in the beginning, it is the purpose of this paper to emphasize the fact that the primary lesion with the regional lymphadenopathy does not comprise the entire clinical picture of tularemia, however frequent this may be. Subcutaneous, lymphangitic nodules, which are usually firm, movable and non-tender, were observed on the arm or forearm along the route of lymphatic drainage in 8.0 per cent of our patients and in a few cases proceeded to suppuration. Such lesions resemble closely those of sporotrichosis.

In 8.44 per cent of our group, a skin eruption occurred and was variable in type. In about half of our patients the eruption was diffuse and papular, although a papulovesicular eruption has been described. In others it was in the form of erythema nodosum. We may point out that the cutaneous eruption usually developed during the second or third week of the illness and persisted for a few days to three or more weeks. It was generally bilateral, symmetrically distributed and appeared most prominently on the arms and neck. Herpes labialis was observed in a few cases.

Cough is observed frequently during the first two or three weeks of illness. Pneumonia developed in 30 patients of our group, an incidence of 13.33 per cent. Seven patients likewise developed pleural effusions. We could fulfill the criteria for tularemic pneumonia in only 21 of these 30 patients, an incidence of 9.33 per cent, the remaining cases resulting from secondary infection. These have been reviewed elsewhere.[4] We may say, however, that the pneumonic process may be described as a hematogenous bronchopneumonia which usually begins at the hilus and extends peripherally producing confluence and variable pleural reaction. The symptoms and physical findings will accordingly be variable and at the beginning may be absent. As one would expect, pneumonic involvement is more frequent in the typhoidal forms of the disease but every patient with tularemia should be carefully observed for the possible development of pneumonia. The mortality rate for our patients with tularemic pneumonia was 57 per cent, thus indicating the seriousness of this complication.

Stupor, meningismus, stiffness of the neck and other evidences of central nervous system involvement may occasionally occur. One of our patients was found to have tularemic meningitis[5] upon autopsy.

Venous thromboses occurred in but three patients, the femoral vein being involved twice and the antecubital vein once. Sev-

eral other conditions were found to be associated with the disease but they did not influence nor were they affected by the tularemic infection.

Course. The course of the illness is chronic and prolonged. Fever occurs in all patients though it may be low grade, intermittent from the onset, and lasts but a few days. In the majority of cases, however, the febrile episodes last for three to four weeks and during this time the constitutional symptoms of malaise, chilliness, muscular weakness, prostration, profuse sweating, anorexia, nausea and vomiting and loss of weight may be quite variable. At best it is rare for the patient to return to work before the second month. In a few of our patients, malaise, weakness, fatigue and lack of a sense of well being persisted upwards to a year. As a consequence the duration of hospitalization in these patients is of considerable importance. The average for our group was 22.35 days and in one patient 144 days of hospitalization were required. Prolongation of illness beyond the periods described above may result from a variety of factors such as delayed suppuration of the buboes, bronchopneumonic involvement and sequelae, and tularemic infection of serous cavities.

The seriousness of tularemic infections has not been appreciated to the degree that they deserve. The mortality rate for our series of 225 patients was 7.55 per cent. It is our impression that negroes manifest less resistance to tularemic infections than do white patients, particularly to the pneumonic forms of the disease. This is borne out by the observation that 12 of 93 negroes (12.9 per cent) died from tularemia as compared to only five of 132 white patients (3.8 per cent). Whether such racial susceptibility may be related to diet or nutrition, environment, lower standards of living or other factors controlling immunity we are unable to state.

The chief cause of death in most of the patients may apparently be attributed to septicemia and toxemia from tularemia. Thus pneumonia was the chief lesion, although meningitis and other findings contributed to the death of one or more patients. The deaths of three of the patients in our series were related to complications arising from therapy: a blood transfusion reaction in one patient, difficulty in controlling diabetes mellitus in another, and a malignant neutropenia following sulfonamide therapy in another. Autopsy of more than half of the patients who died verified the widespread tularemic involvement as evidenced by pathologic lesions in the skin, lungs, liver, spleen and lymph nodes. As one would expect, the mortality rate for each of the four clinical types of tularemia varies considerably: 4.4 per cent for ulceroglandular type, 4.35 per cent for glandular, 14.3 per cent for oculoglandular, and 50 per cent for the typical form of the disease. Death may appear at any phase of the illness, the average duration of disease for our group that died being eight days before death with the shortest duration being 36 hours and the longest 167 days (an instance of chronic progressive tularemic infection).

We have no data in our series concerning relapses of tularemia. The literature records relapses rarely; when they occur, they appear within eight months to two years following the first attack. A second relapse is extremely uncommon. Equally rare is the condition of chronic tularemic infection in which the clinical course is very disabling and characterized by malaise, weakness, loss of weight, intermittent fever, mental as well as physical depression, enlargement of previously affected lymph nodes and spleen, and downhill course. In the majority of patients, clinical recovery confers permanent immunity. Foshay[3] states that subclinical or inapparent tularemic infection has never been demonstrated.

Complications and Sequelae. These are not common. We have already alluded to the seriousness and frequency of septicemia during the course of the tularemic infections. Although known to occur during the first week of illness, it more commonly develops toward the end of the second week and beginning of the third week. The onset of septicemia may be heralded by the fol-

lowing signs: (1) sustained high fever following a period of intermittent variations; (2) gradually deepening coma and stupor; and (3) progressive and at times rapid enlargement of the liver and spleen. The prognosis of tularemic septicemia is admittedly grave.

The sequelae of pleuropulmonary tularemia are bronchiectasis, extensive pulmonary fibrosis, empyema and lung abscess. Rarely seen but recorded are instances of pericarditis, peritonitis, meningitis,[5] encephalitis, enteritis, venous thromboses and thrombophlebitis. More common but less serious are occasional recurrences of lymph node enlargement from one to several years following apparent recovery and in which suppuration of the involved glands occurs in more than half of the cases.

LABORATORY DIAGNOSIS

Clinical diagnosis of the ulceroglandular and glandular types of the disease is not usually difficult if the physician obtains a history of recent contact with a wild rodent, tick bite, or fly bite. The typhoidal form of the disease, however, demands recourse to the laboratory for confirmation of the diagnosis and frequently for establishment. There, too, the physician will do well to recall the geographic distribution and seasonal incidence of tularemia and to interrogate for possible animal contact. We have occasionally been gratified to suspect and later confirm the diagnosis of tularemia in atypical bronchopneumonia by obtaining a history of contact and finding an indolent ulcer on the hand during the course of the physical examination.

Blood Changes. There are no characteristic changes in the blood. Most of our patients revealed a mild secondary anemia, the red cell count averaging from 3,500,000 to 4,000,000 per cubic millimeter. The average leukocyte count was 11,200 with a differential count averaging 70 per cent polymorphonuclear leukocytes, 25 per cent lymphocytes, and 5 per cent monocytes. It is interesting to note that five of our patients had white cell counts of less than 5,000 cells whereas another patient with typhoidal tularemia had a count of 90,000 on admission.

Urinalysis. A transient albuminuria was noted in a few cases and was related to the febrile course.

Agglutinations. The agglutination test for tularemia is extremely satisfactory. Agglutinins never appear before the eighth or ninth day but thereafter a progressive increase in titers occurs up to the sixth week of the illness or beyond. A titer of 1:20 is suspicious and 1:80 is diagnostic. More significant is a progressive or rising agglutinin titer. Cross agglutinations with Brucella may occur; the highest titer usually indicates the primary disease. Agglutinins may persist indefinitely in the serum of patients, possibly for life. One of our patients had a titer of 1:320 seven years following recovery.

Skin Test. The intradermal injection of a killed suspension of *B. tularense* provokes a cutaneous response similar to the tuberculin test. The test is positive in about 90 per cent of patients sometime during the first week of the disease and thus serves to establish a tentative diagnosis before the agglutination test becomes positive. Local necrosis at the site of injection has occurred in about 25 per cent of our patients. Like the agglutination test, the skin test is positive for many years.

Bacteriologic Diagnosis. Examination of stained smears of exudates from the primary lesions, lymph nodes, sputum or serous cavities is virtually useless. A bacteriologic diagnosis is possible during the stage of bacteriemia or septicemia by direct inoculation of the patient's blood on fresh blood-glucose-cystine agar. Even better is the inoculation of the infected secretions or body fluids into guinea pigs or other rodents and then cultivation of the organism from the rodent's heart blood at autopsy. It should be pointed out here that laboratory cultivation of *B. tularense* is extremely dangerous for non-immune personnel.

TREATMENT

As in any acute febrile disease with severe constitutional reactions, symptomatic and supportive care of the patient assume an important part of the therapeutic re-

gime. Although classed as a communicable disease and considered reportable in most cities and states, rigid isolation precautions for tularemia are not necessary as man to man transmission has occurred very rarely.

Medical Regimen. No form of therapy has proved to be consistently beneficial in our experience. In line with other investigators, our series of patients were subjected to a wide variety of therapeutic agents: sulfarsphenamine, neoarsphenamine, roentgen ray, infra-red light, specific serum, human convalescent serum, acriflavine, and the sulfonamides. None of these agents can be said to effect consistent or marked influence upon the course of the disease. Available evidence, though meager, suggests that penicillin likewise has no particular value in the treatment of tularemic infections. Recently Heilman[6] has reported that streptomycin is of value in experimental tularemia. This deserves clinical investigation.

The whole subject of the therapy of tularemic infections has been reviewed recently by Bell and Kahn[7]. These investigators conducted careful animal experiments upon the therapeutic efficacy of the following drugs: sulfanilamide, sulfadiazine, sulfamerazine, acriflavine, metaphen, iodide and bismuth (iodo-bismitol with saligenin), arsenic and bismuth (solution of bismuth subgallate and sodium para-aminophenyl arsonate), trivalent arsenic alone (mapharsen), antimony (stibophen), penicillin and hyperimmune equine antitularemic serum. With the possible exception of penicillin, they concluded that none of the above mentioned drugs possesses any therapeutic value in tularemic infections.

Local Applications. The primary lesions and suppurative lymph nodes have been treated with various local applications: 2 per cent saline compresses, boric acid soaks, magnesium sulfate soaks, roentgen ray, infra-red, acriflavine, 5 per cent sulfathiazole ointment, weakly saturated solution of commercial urea, and occasionally incision. We conclude from our experience that simple sterile dressings kept moistened with boric acid or saturated solution of magnesium sulfate are as effective as any available regimen in affording relief of pain from the buboes. We wish to emphasize that it is our practice never to incise the inflamed lymph nodes until suppuration has occurred and rupture is imminent.

For the eye lesions, local applications of various types have likewise been tried: wet boric acid compresses, argyrol, homatropine, hot saline soaks, 5 per cent solution of sulfathiazole, and hot applications of half saturated aqueous magnesium sulfate. We firmly believe that frequent lavage of the conjunctival sac with warm boric acid-saline solution is of benefit. External, soothing applications to the eye should probably be warm, sterile, and non-injurious; in that regard, the half saturated aqueous magnesium sulfate solution is as good as any agent.

Prevention. General awareness by the lay population of the danger of contracting tularemia in handling and skinning rabbits and other game should be developed not only in hunters but also in meat handlers, butchers and housewives. The use of rubber gloves, although not ideal, will lead to a marked reduction of infection. Thorough cooking of all meat will eliminate the dangers of infection by ingestion. There is no satisfactory method for the prevention of tick and fly bites. Foshay and his associates[8] have reported favorably on the use of a vaccine in the prophylaxis of tularemia. They believe that the disease is not only prevented in a large percentage of instances but greatly modified as well. Laboratory personnel are particularly subject to tularemic infections and should review the prophylaxis and handling of tularemic cultures, tissues and specimens in meticulous detail.

SUMMARY

We have sought to clarify herein the salient points in the clinical recognition and management of tularemia based upon our studies of 225 cases. Attention has been directed to the widespread distribution of the disease, the large number of animal contacts, the atypical expressions,

the case fatality rate (7.55 per cent), and the methods of diagnosis and treatment. We have also pointed out that the mortality rate is four times greater among negroes than among white patients. The prolonged morbidity and high mortality of tularemia stamp it as of special importance among infectious diseases. It is regrettable that a satisfactory therapeutic agent has not yet been found.

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NEW ORLEANS

Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor-in-Chief, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

VICTORY!

The war ceased abruptly on August 14. It hardly seems possible after nearly four years of war-time regulations that hostilities have stopped. The transition period from war to peace appears at the present time to be more difficult than the change from peace to war. It took the United States several years before they really began active participation in the practically world-wide war and during these two years adjustments to war could be made more deliberately than apparently it will be possible to do so for peace, speaking of the over-all picture.

How the cessation of hostilities will affect the medical profession no one, certainly at the present time, is in a position to say. The physicians in the Army are anxious to go back to civil practice and certainly the doctors who have stayed at home are pretty well worn out because of their additional responsibilities and tremendous increase in the amount of work they have been called upon to do. How soon medical officers will be discharged is a question. Certainly it does not look as if there would be an immediate return of a very great number of physicians to civil life. The large number of casualties who will require months and years of treatment will have to be taken care of by civilian doctors who are in the Army, more particularly those who have specialized training. It will be hard luck for these doctors if they have to be kept in the service for months or even years. It is earnestly hoped that these men will be but few in number and will be willing to answer the call of duty cheerfully.

We can all thank God that the war is over, a war which from the beginning was senseless and useless. The United States emerges as the greatest world power. We and Russia dominate the world and our responsibilities should be world-wide and not limited to this loved country of ours nor to this continent.

SOCIALIZED MEDICINE

There has been considerable activity during the summer both by the protagonists and antagonists of the Wagner-Murray-Dingell bills. These bills have been discussed over the radio, presented in one of the radio forums, have been written up in the newspapers, and medical societies throughout the United States have expressed themselves against any form of socialized medicine. The American Medical Association has drawn up fourteen points which bear reading by the individual members of state, county and parish medical societies. The Physicians Committee for the Extension of Medical Service has sent out

much material, including a communication to officers of all medical societies, saying that if the bills are enacted into law the private practice of medicine in the United States would be destroyed. They have also sent out copies of newspaper advertisements, plates of which are available without cost and which it is suggested should be placed in newspapers under the sponsorship of local medical societies, hospital groups, drug stores and similar groups and organizations.

All these efforts show that the leaders of medicine are doing their best to educate the public to the evils of regimented medical practice. The sad and unfortunate thing about these efforts to counteract socialization of medicine is the general apathy of individual members of the profession. There are a few men who are doing their best to counteract governmental interference in medical practice but they are few and far between. Most of the physicians of this country are, it is true, very busy but they could exhibit and should show much greater interest in the socialistic program of the Wagner-Murray-Dingell bills. The attitude seems to be by the profession that the officers of their societies should do all the work. In many other things that are unpleasant, physicians, as do other people, have a habit of not looking at disagreeable things squarely in the face, assuming that such and such a proposal will never come about, or it will not bother them in their life time, they take an attitude which is, to say the best merely neutral. Each and every individual doctor in this country who is a member of organized medicine should do his best to counteract the arguments that are advanced by the people in favor of socialized medicine. They can talk to their clientele, talk or write to their Congressmen and they can appear at public meetings to point out the inherent evils of medical regimentation. It is up to each and every man who is sincerely against the proposals being made in Congress to express himself with vigor and enthusiastically.

THE IMMEDIATE TREATMENT OF CORONARY OCCLUSION

The immediate treatment of coronary occlusion, or myocardial infarction, is one of the medical emergencies which requires prompt and vigorous treatment. It is a condition which is associated with pain and with shock and it is these two indications which first must be combated before giving serious thought, in most instances, to the state of the heart. Pain is an almost inevitable and invariable concomitant of occlusion and as it intensifies and prolongs shock it must be overcome promptly. Morphine is the drug of choice because it is more than likely to relieve the pain promptly than any other analgesic. It should be given in doses sufficient to bring about relief of the cardiac agony. Morphine is by no means the ideal drug because of its vasoconstricting effects on the coronary circulation. Other analgesics have been advocated from time to time such as dilaudid or papaverine. It might be possible in many instances to use papaverine if the pain is not too severe, giving it in doses of 30-80 mg., repeating it if necessary. With morphine, atropine should always be given as it counteracts the effects of this drug on the coronary circulation. As a matter of fact it might be advisable to give atropine more or less routinely with the other two pain remedying drugs.

Circulatory failure which is associated with the shock syndrome is best met by the administration of adrenalin. With shock there is associated severe sweating with loss of fluids and electrolytes. To combat these two indications for therapy, normal salt solution may be given subcutaneously, or 5 per cent glucose in saline solution may be given slowly into the vein. Oxygen should be started at once and immediately. Gilbert insists that it should be employed regularly in all instances but in some of the questionable mild attacks it is doubtful if it is necessary to resort to this therapeutic procedure, unless readily accessible.

Some years ago Levine suggested quinidine to prevent paroxysmal ventricular tachycardia which occurs in 3 per cent of

all cases and which may be followed by ventricular fibrillation and death. This form of therapy has not received universal approval and as a matter of fact Levine now apparently recommends it only when there is a very rapid heart rate associated with a considerable drop in blood pressure.

Cardiologists generally advise against the administration of digitalis. It increases the work of the heart and may bring about rupture of a soft infarct; it produces coronary constriction and it is presumed to predispose to paroxysmal ventricular tachycardia. Furthermore, most patients who have coronary occlusion do not have signs of heart failure for the first week or ten days or two weeks after they have their coronary attack. If a patient does exhibit definite signs of failure either of the right heart with a swollen, tender liver, or of the left heart with very marked dyspnea or if auricular fibrillation develops then the doctor should make use of digitalis.

The immediate and prompt and proper treatment of coronary occlusion is often not only life saving at the moment but may prevent heart failure and death in the immediate future. If a patient can be successfully carried over the first phases of his disease it may mean subsequent years of a fruitful and constructive life.

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ORGANIZATION SECTION

The Executive Committee dedicates this page to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

CURRENT TRENDS IN MEDICINE

In the July 28, 1945 issue of the American Medical Association Journal there appeared a letter signed by Dr. L. G. Rowntree, which read as follows:

"To the Editor: In retiring from Selective Service, after serving as chief of the Medical Division during the last four and one-half years, I have received literally dozens of letters complimenting us on what has been accomplished. Most of these letters, I note, came from physicians. In reading them and in thinking over the situation, it seems to me that the shoe really should be placed on the other foot. Hence I am writing to express the gratitude of the Medical Division of Selective Service to the 35,000 doctors and 10,000 dentists who have contributed so liberally of their time, effort and skill in handling the medical problems of the registrants.

"No other group of people in the nation have given more liberally of themselves in the war than have these professional people. That was the opinion expressed by the late President Roosevelt at one of his wartime conferences.

"The Medical Division of Selective Service in National Headquarters in Washington has enjoyed the complete support of the director, Major Gen. Lewis B. Hershey. It has had also the strong support and superlative leadership from the three Surgeons General of the Army, Navy and Public Health Service, from all the state headquarters and especially from all the state medical officers. The physicians attached to the 6,443 local boards, the consultants on the 660 medical advisory boards supplied all the demands made on them. They met fully and completely the nation's needs in the procurement of manpower for the fighting forces.

"Such service should not be taken for granted nor should it be passed over without acknowledgment. Medicine has done a grand job in this war in Selective Service as well as in the Medical Corps of the Army, Navy and Public Health Service.

"In leaving Selective Service I wish to express to all the civilian physicians and dentists in the Selective Service System my personal appreciation and that of the Medical Division at National Headquarters for

the wholehearted service they have rendered throughout the last four and one-half years.

L. G. Rowntree, M. D.
Selective Service System
Washington, D. C."

We know that the many physicians in Louisiana who served on the examining boards for the Army, Navy, and Public Health Service will be gratified to learn that their services gratis to the government have not really been forgotten. While this comes as a late citation, in our opinion it is justly merited. It is too bad that as Colonel Rowntree, appearing before the Pepper Sub-Committee on Education and Labor and discussing hospital expansion, could not have given to this committee such a wonderful recommendation for the medical profession. When he appeared before this committee his presentation could not have been taken as a compliment to the accomplishments of the medical profession in relation to the physical defects that occurred as a result of the rejections by Selective Service. It is very interesting to note that from these hearings and study by the sub-committee many bills have been proposed and introduced in Congress proposing to correct the so-called defects and the faulty distribution of medical service. Many astute political students are of the opinion that the Wagner Bill is a true camouflage; that the proposals in Senate Bill 191 and Senate Bill 1318 are in reality a more forcible regimentation than in Senate Bill 1050. Senate Bill 1050 contains the setup for the expansion of hospitals, maternity and infant care as Senate Bill 191 and Senate Bill 1318. This represents a high form of political ingenuity.

Senate Bill 191, known as the hospitalization bill of which we have spoken very frequently in these columns, is the outgrowth of this study. However, they have also taken a new step which is not at all surprising with the introduction of Senate Bill 1318. This same Senator Pepper introduced for himself and other members of the Committee on Education and Labor the bill whose title reads "To provide for the general welfare by enabling the several States to make more adequate provision for the health and welfare of mothers and children and for services to crippled children, and for other purposes." When Senator Pepper introduced this bill, he described it as "a modest beginning." He said, in part:

"Medical care and health supervision of children is costly in dollars. Reliable authorities estimate it comes to somewhere in the range of $25 to $40 a year for each child in the United States. With 40,000,000 children under 18 that represents a total of at least $1,000,000,000 for the country. A Federal appropriation of $75,000,000 for maternal and child health and for crippled children for a year cannot go very far in meeting these all-over health needs of children. Even if it were divided with mathematical precision among all children, it would come to less than $2 a child. Of course, it will not be so divided, but that kind of calculation helps to indicate the modesty of the approach in this bill.

"If we were at peace, the sums called for now would appear inadequate in the extreme. But we are still at war. We can only inch ahead at this time. Many doctors, nurses, and other trained personnel who could help us expand our services for children are in the armed forces. When they are released from service, they will help us. New personnel—and we will need a large expansion — cannot be trained overnight. Health and welfare services, if they are any good, are manned by workers with years of specialized training and experience behind them. The greatly expanded demand for health and social personnel and the greatly enlarged opportunity for the training of such workers, created by this measure, will make a constructive contribution to national policy of peacetime full employment.

"Obviously, a Nation-wide child-health and child-welfare program is not something that can be created in a year. For that we must have time to develop services, train personnel, develop facilities, conduct research and demonstrations, and educate parents in the use of facilities and in the application of expanding scientific knowl-

edge. The authorizations for appropriations that we are suggesting for this year will give us a fair start toward our objective, though it will be only a beginning."

Here we have in its crude form a manifestation to expand and to extend the emergency maternity and infant care program and to make it a permanent feature in their postwar planning. Of course we all should remember that the contract we had with the Children's Bureau in this state providing for the emergency maternal and infant care program was entered into after a great deal of pressure and with some stringent reservations. One of the most important of these reservations was that the program should be only for the duration of the war. Now they are preparing to make this service a permanent one for the civilian population, using as an argument that the system has proved to be beyond their expectations in success and has materially reduced the maternal infant mortality rate in this country. We are at present trying to get some statistical data from our state in regard to this latter claim. Therefore, it behooves every member of our State Society to write to his representatives and secure a copy of this bill and study closely its implications. If you would refer to the August 11 issue of the American Medical Association Journal, pages 1101-1112, you will find some valuable information on this subject. After you have given the consideration that is required, you should write or contact your representatives and senators and give them your opinion concerning same. Congress is now in session. The opportunity may be presented for a personal contact with these representatives. A personal contact will be much more effective than any other method. However, if this is not possible, write them your opinion concerning same.

Likewise, you should study Senate Bill 1050, known as the Wagner-Murray-Dingell Bill, and contact or write your representatives and senators concerning this bill. Also you should have your friends, relatives, fellow club members, and other organizations do likewise. Look at the daily press today and you will find quarter page advertisements sponsored by some commercial organizations supporting the wonderful features of social security. You should not forget that the present Wagner-Murray-Dingell Bill is an amendment to the social security act. It is evident that an extensive form of advertising is now being carried on with the full knowledge of those who are sponsoring the Wagner-Murray-Dingell Bill. Now is the opportune time for us to try to counteract these nefarious activities. Unquestionably, very shortly hearings will be had on both of these bills. Appropriate medical representation undoubtedly will be present at these hearings. However, the ground work can be forcibly perfected by your timely cooperation and assistance. Do it now!

Our Committee on Postwar Medical Planning, as well as the profession at large, is greatly concerned with the official plan for the discharge of medical officers from the military forces. For information on this important subject, please refer to the August 11 issue of the American Medical Association Journal, pages 1102-1104. While our medical profession is preparing itself to aid and assist these returning medical officers, it is not clearly understood from these official reports just how rapidly they will be discharged from service. In the meantime, we are exerting every effort possible to make this transformation easy and comfortable for them.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Sept. 12. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Sept. 13. Clinico-pathologic Conference, Touro Infirmary, 12:00 noon.

Sept. 19. The Orleans Society of X-Ray Technicians, 7:30 p. m.

Sept. 26. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Sept. 27. Clinico-pathologic Conference, Touro

Infirmary, 12:00 noon.
DePaul Sanitarium Staff, 8 p. m.
Sept. 28. L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Oct. 1. Board of Directors, Orleans Parish Medical Society, 8 p. m.
Oct. 2. Eye, Ear, Nose and Throat Staff, 8 p. m.
Oct. 3. Mercy Hospital Staff, 8 p. m.
Oct. 4. Executive Committee, Baptist Hospital, 8 p. m.
Oct. 8. Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
Oct. 10. Woman's Auxiliary, Orleans Club, 3 p. m.
Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Oct. 11. Clinico-pathologic Conference, Touro Infirmary, 12:00 noon.

There was no meeting during the month of September as the Society was in recess; meetings will resume on October 8. Members of the Touro Infirmary staff will present the program at the October meetings.

NEWS ITEMS

Dr. Joseph Menendez recently addressed the Quota Club.

Lt. James W. Tedder was the recipient of the Bronze Star Medal award with citation for heroic and meritorious service to survivors of a torpedoed, sunken ship in Lingayen Gulf, during the invasion of Luzon in the Philippines last January. The presentation was made during personnel inspection 7 July by Captain H. E. Jenkins, Senior Medical Officer.

MONTHLY STATISTICAL REPORT OF NEW ORLEANS, JULY, 1945

Estimated Population as of July 1, 1944

White		391,000
Colored		169,000
Total		560,000
Total Deaths, All Causes		538
White	330	
Colored	208	
Resident Deaths, All Causes		418
White	250	
Colored	168	

DEATH RATES

(Per 1000 per annum for the month)

	All Deaths	Non-residents excluded
White	10.2	7.6
Colored	14.8	11.9
Total	11.5	8.9

Total Births Recorded		1,244
White	799	
Colored	445	
Resident Births		962
White	609	
Colored	353	

BIRTH RATES

(Per 1000 per annum for the month)

	All Births	Non-residents excluded
White	24.5	18.7
Colored	31.6	25.0
Total	26.6	20.6

Max M. Green, M. D.,
Secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

STATE MEETING TO BE HELD IN APRIL 1946

At the meeting of the Executive Committee of the State Medical Society held Saturday, August 25, it was decided to hold the annual state meeting on April 29, 30, and May 1, the place of meeting to be decided upon later. The details of the Executive Committee meeting will be published next month.

64th GENERAL HOSPITAL HONORED

Word has been received by Dr. Urban Maes, Director and Professor of Surgery, Louisiana State University School of Medicine, that the Meritorious Service Unit Plaque was presented to the 64th Military General Hospital This unit is sponsored by the L. S. U. School of Medicine and was organized by Dr. Maes. Its medical officers

are members of the faculty and alumni of the school. The Commanding General of the Peninsular Base Section presented the plaque at retreat ceremonies July 25, 1945, at Livorno, Italy. The citation entitles members of the unit to wear the wreath on the right forearm sleeve. The citation reads as follows:

CITATION FOR MERITORIOUS SERVICE UNIT PLAQUE

The 64th General Hospital for superior performance of exceptionally difficult tasks and outstanding devotion to duty in the Peninsular Base Section for the period 1 August 1944 to 1 October 1944. This medical unit was the first hospital in line of evacuation of casualties from fighting in the Arno River sector. This hospital quickly adapted itself to field hospital techniques. From 1 August 1944 to 1 October 1944 the professional service of the hospital functioned on a 24-hour basis. The superior care of medical patients and battle casualties was consistent with the unselfish ideals of the medical service of the United States Army. With rigid discipline, full knowledge of and devotion to its mission, the 64th General Hospital upheld the highest traditions of the United States and of the allied nations.

BASE HOSPITAL NO. 24

The following excerpt from a letter from one of the medical officers connected with Base Hospital No. 24 gives some information as to the status of this organization. "As you know, the 24th has all but disintegrated. We closed the hospital in Florence on May 23 after nine months of busy and interesting work in that location. Shortly thereafter re-deployment began and most of our officers and nurses with points below 80 were transferred to Pacific bound units and others to occupation hospitals. Three-fourths of the men in the medical detachment have been transferred. The medical service lost John Archinard, Buddy Matthews, Bob Snavely, Pierre Poole and Tom Weiss who were all transferred to Pacific bound evacuation and station hospitals, and in fact have already left for the Pacific. The surgical service lost Frank Cox, Sidney Charbonnet, Joe Sabatier, Morty Silvey, Dick Vincent, Homer Parnell and Phil Trout. All the dentists have left except Charles Rossner. Most of our nurses have been transferred. As you see, the 24th is now nothing but a broken down old steleton, war torn and weary, and badly in need of physical and spiritual rehabilitation. Those of us who remain do not know what the future holds in store for us but rumor has it that the 24th will be leaving for the states sometime during the next two months and will be in a strategic reserve pool awaiting new assignments."

MAJOR KAGY FREED

The many friends and classmates of Major Edwin S. Kagy, Tulane Medical School 1934, were delighted to read that the Major was freed from a camp at Yokohama after having been a prisoner of war in Japan since the fall of Corregidor. According to the newspaper reports, Major Kagy had been allowed to practice among the prisoners of war in the camp in which he was located.

Major Kagy's wife, the former Mary Crossen, was assistant secretary-treasurer of the Louisiana State Medical Society before her marriage. Mrs. Kagy is known to many of the doctors of the state. After Major Kagy's capture, she was good enough to resume work with the Louisiana State Medical Society and the Journal.

DR. W. C. SMITH NEW DEAN

The innumerable friends of Dr. Wilbur C. Smith have heard with pleasure that this well known teacher of anatomy at Tulane University School of Medicine has been elevated to the position of Dean of the Louisiana State University Medical School. Dr. Smith has taught anatomy at Tulane for twenty-seven years. While at Tulane Dr. Smith was identified for some twenty-three years with the department of physical education. At the present time he is president of the National Collegiate Athletic Association. Every one will wish him success in his new undertaking.

Dr. Hiram W. Kostmayer, Dean of the Tulane School of Medicine, announces that Dr. William B. Wendel has succeeded Dr. Sydney Bliss as professor and head of the department of biochemistry at Tulane. Dr. Wendel is a graduate of Emory University, he did postgraduate work at the University of Chicago, University of Tennessee, and Washington University. He was associate professor of chemistry at the University of Tennessee before coming to Tulane.

COMMENCEMENT EXERCISES OF TULANE UNIVERSITY MEDICAL SCHOOL

At the annual Graduation Exercises held August 14, 1945, the graduating class was addressed by Dr. Emil Novak, the distinguished Baltimore gynecologist. Following the address, commissions were awarded in the Medical Corps of the Army and in the Naval Reserve to those members of the class who were educated under the Student Training Program. This class was the first to graduate that had completed this program, taking three years continuously instead of the usual four years.

Three Paul Tulane Fellows were selected from the Senior class, these men being Drs. Thomas G. Baffes, John Jacob Baehr and Rodney Clifton Jung. Dr. Walter O. Moss of Lake Charles, vice-president of the Tulane Alumni Association, spoke in behalf of the organization to the members of the graduating class. The invocation and benediction were pronounced by the Reverend Lemuel

Davis Haughton whose son was one of the graduates.

IVY DAY AT TULANE

The graduating class of Tulane celebrated Ivy Day on Monday, August 13, on the uptown campus of the University. The Ivy Day program was presided over by Dr. Rodney C. Jung. The annual address was delivered by Dr. Charles E. Dunlap, Associate Professor of Pathology and Bacteriology.

The Walter Reed Memorial Medal, given by the Louisiana State Medical Society to the graduate presenting the best thesis either on tropical or preventive medicine, was presented by Dr. A. V. Friedrichs, chairman of the Walter Reed Memorial Committee, to Dr. Benjamin T. Galloway. In this presentation Dr. Friedrichs mentioned that Dr. Iwao Uyeda received honorable mention for his excellent thesis. Other awards included the Querens-Rives-Shore Award given to Dr. A. V. N. Mortensen, with honorable mention to Dr. A. Norman Evans and Dr. William D. Franklin. The Sidney K. Simon Prize was awarded to Dr. Leo E. Johns, with honorable mention to Dr. George E. McCaskey. The Professor of Medicine Award was divided between Dr. William T. Newsom and Dr. Thomas C. Donald. The Isadore Dyer Medal for the highest grades obtained in four years of medical school was awarded to Dr. Thomas G. Baffes. The Jacob C. Geiger Medal was given to Dr. Mitchell Ede.

The ivy was planted by Drs. Rodney C Jung, John J. Baehr and Luther T. Tyler.

THE U. S. PHARMACOPOEIA

The U. S. Pharmacopoeia observes its 125th anniversary this year. For one hundred and twenty-five years, the USP has hewn to its purpose "to provide standards for drugs and medicines of therapeutic usefulness or pharmaceutic necessity, sufficiently used in medical practice within the United States or its possessions; to lay down tests for the identity, quality, and purity of these; to insure, so far as practicable, uniformity in physical properties and active constituents."

WAR-TIME GRADUATE MEDICAL MEETINGS

The war-time graduate medical meetings sponsored by the American Medical Association, the American College of Physicians and the American College of Surgeons will probably not discontinue their activities until mobilization has been carried further than it is at the present time. One of these meetings will be held at the DeWitt General Hospial, Auburn, California on September 14. Dr. Alton Ochsner will participate in a peptic ulcer symposium as well as presenting a paper on surgical treatment of empyema. Lt. Col. Ambrose Storck will take part in a symposium on the care of paraplegic patients and will also appear on the program in a symposium on vascular disease.

Colonel Roy H. Turner, M. C., of New Orleans, formerly Communicable Disease Treatment Branch, Medical Consultants Division, has been assigned overseas.

The School of Medicine, University of Pittsburgh, offers an Orientation Course in Clinical Allergy, under the sponsorship of The American Academy of Allergy, for five days, October 1 to 5, 1945, inclusive, at the School on Bayard Street, Pittsburgh, Pennsylvania. Fee $40.00; for veterans, service men, and residents $10.00. Registration for evening round table conferences only by special arrangements.

Inquiries should be addressed to William S. McEllroy, M. D., Dean, School of Medicine, University of Pittsburgh, Pittsburgh 13, Pennsylvania.

THE NEED FOR PHYSICIANS

The following release has been cleared through the facilities of the Office of War Information. It indicates that others besides the medical profession realize that there will be a critical shortage of physicians and dentists, and unless some serious effort is made to supply the deficiency which will exist, the medical schools will not have a sufficient number of students to turn out the much needed doctors. The release in part reads:

With the nation facing a serious shortage of doctors and dentists in the postwar period, Paul V. McNutt, chairman of the War Manpower Commission, today announced plans to enroll immediately 12,000 students for medical, dental, premedical and pre-dental school courses beginning this fall from among veterans now being discharged from the armed services.

Mr. McNutt said surveys show that even if medical school enrollments, admissions and graduations had been maintained at levels prevailing before recent changes in Selective Service policies there still would have been a postwar shortage of thousands of doctors and an even severer shortage of dentists.

"Unless we move quickly and successfully to wipe out the gaps torn in the ranks of the medical and dental professions by the exigencies of war, America will be denied essential security in terms of national health," Mr. McNutt said.

The situation is so severe that some medical schools will not fill any substantial part of their first-year classes beginning next month, he added.

With a goal of 8,000 students for medical and pre-medical schools and 4,000 for dental and predental schools, the War Manpower Commission, with the cooperation of the Army, the Navy, the Veterans Administration and the medical and dental professions, will place the story of the op-

portunities in the medical and dental professions before men in the armed forces who are scheduled for discharge in the near future.

At all separation centers the most likely candidates for medical and dental and pre-medical and pre-dental schools will be screened on the basis of their qualifications and expressed interest.

While nearly 40 per cent of the active practicing physicians of the United States are now in the armed forces, the chief concern at present is not only the wartime supply of physicians and dentists for civilians, but also the supply of physicians and dentists for veterans and the peacetime armed services. As a consequence of the war, Mr. McNutt said, many more physicians will be needed than were available before the war. Present estimates are that a peacetime army will require 10,000 physicians, a peacetime navy about 5,000 and the Veterans' Administration about 15,000.

Furthermore, the widespread destruction of hospitals and medical schools and physician casualties in the liberated countries of Europe and China will require the aid of unknown numbers of physicians from the United States, he said. The number of physicians in the armed forces of the United States killed or incapacitated by the war, and requiring replacement, together with the high mortality among over-worked physicians who remained at home during the war are other known factors whose effects cannot be accurately predicted.

On the basis of the information now available, Mr. McNutt said, it is conservatively estimated that about 35,000 additional doctors will be needed. But a survey by the American Council on Education and the Office of Scientific Personnel of the National Research Council has indicated that less than half of this need will be met.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the week ending July 7 was one of the most remarkable ones that has ever emanated from the State Board of Health. There were only two diseases listed in numbers greater than 10, namely, malaria with 21 cases and measles with 14. As 13 of these cases of malaria were contracted outside of Continental United States, it may be said truthfully that measles was the only reportable disease which carried in numbers over 10. There were two cases of poliomyelitis reported this week, and seven cases of typhus fever. For the week which closed July 14, again the reportable diseases were distinctly small. There were listed 64 cases of pulmonary tuberculosis, 25 of malaria, 17 of unclassified pneumonia, 15 of typhus fever, 13 of septic sore throat, and 10 of diphtheria. The poliomyelitis cases numbered only one. The typhus fever patients, with Lafayette and Iberia Parishes each reporting four cases, came from other parishes in the southern part of the state. For the week ending July 21, pulmonary tuberculosis again led all other reportable diseases with 58 cases. Other diseases occuring in double figures included 16 cases of malaria, 10 contracted outside the United States, 11 cases of typhus fever, and 10 of diphtheria. The four cases of poliomyelitis that were reported this week came two from Caddo and two from Bossier Parish. The typhus fever patients were found four in Iberia Parish, three each in Orleans and Avoyelles, and one in St. James Parish.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week which closed July 14 there were listed a remarkably small number of deaths in the City of New Orleans, there being only 99 people expiring in this particular week, as contrasted with a three-year average of 132. Of these people dying in the city, 62 were white, 37 were colored, and 10 were in infants under one year of age, divided equally between the two races. There was a very sharp increase the following week, evidently all the very sick people of the previous week hanging on for longer than the few days they should, because there were 44 more deaths this week, 88 among the white population, 55 in the colored, and of these 13 were children under one year of age. For the week which closed July 28, there was quite a sharp decline. Of the 119 people who expired in New Orleans that week 71 were white, 48 colored, and only seven of these were children under one year of age. The deaths in New Orleans for the week of August 4 were almost exactly the same as the previous week, 120 dying this week, divided 70 white and 50 negro. There was a considerable increase in the deaths of children, 18 of the little tots dying in this particular week.

TUBERCULOSIS DEATH RATE IN LOUISIANA

The death rate in Louisiana is among the highest of any state in the United States. It ranks eighth with a death rate of 50.5 per 100,000 population. The death rate for the United States as a whole is 42.6. Arizona, as might be expected because of the large number of people who migrate there who have tuberculosis, leads all states with a rate of 98.2, followed then by Tennessee, New Mexico, Kentucky, Nevada, Maryland and the District of Columbia The rate is lowest in the state of Utah, being 11.2. Kansas, Idaho, Iowa, Nebraska and Wyoming are other states that have a rate under 20. These figures are from the United States Bureau of the Census, June, 1945.

7TH WAR LOAN

The following letter was received from the Treasury Department:

Our gratitude seems small-fry!

It's inadequate for the generosity, for the patriotism, for the effectiveness of publishers' co-

operation during the 7th War Loan.

That's why our simple words of appreciation for your vital war effort support don't measure up to the Treasury's pleasant wish—to tell you how grateful we are. But your magnificent war financing contribution has other rewards: it's a helping hand for the care of our wounded, a strong blow to down any threat of inflation, a kind friend to fulfil hopes for education, homes, security.

Elihu E. Harris,
Chief, Periodicals Section.

BOOK REVIEWS

Intravenous Anesthesia: By R. Charles Adams, M. D., C. M., M. S. (Anes.). N. Y. Paul B. Hoeber, Inc., 1944. pp. 663. Price, $12.00.

Intravenous anesthesia has been the dream of surgeons ever since the discovery of surgical anesthesia. Before the development of the barbiturates, no satisfactory drug was available, though many were tried. Dr. Adams has traced the history of intravenous anesthesia step by step and described each drug that has been tried. He has discussed the advantages and drawbacks of each. A good deal of the book is devoted to the pharmacology of the barbiturates. Pentothal, in particular, is described in detail and emphasis is placed upon the technic of its use in clinical surgery. The subject matter, both the historical and practical aspects, is covered in great detail and supported by an excellent bibliography. The book will be of interest primarily to physicians, devoting their time to anesthesiology, to pharmacologists, and to others who work with depressant drugs. The general practitioner of medicine may find it useful as a reference. The author has done an excellent piece of work with a difficult subject.

JOHN ADRIANI, M. D.

Peripheral Nerve Injuries: By Webb Haymaker, Capt. M. C., A. U. S., and Barnes Woodhall, Maj., M. C., A. U. S. Philadelphia, W. B. Saunders Co. 1945. pp. 227. Price, $4.50.

Presented in the text of this distinguished manual is all of the information that has been established concerning peripheral nerve injuries and new data that have accumulated during the war. It is an appraisal of a wide field of knowledge.

The book is divided into three sections: the first deals with the principles of innervation; the second is concerned with special methods of examination of the patient; the third has to do with the clinical features of plexus and peripheral nerve injuries. At the beginning of each section there are brief remarks intended to indicate the highlights of its contents. There are numerous, excellent photographs, charts, diagrams and illustrations.

While much of the contents of this publication is highly specialized and technical and will be used mostly by neurologists and neurosurgeons it will be of unusual usefulness in the field of general surgery. Its illustrious authors have herein made more than an ordinarily important contribution to medical literature. Their work manifests fine discrimination and judgment in its preparation. It is a vital, timely necessary book.

C. P. MAY, M. D.

The Examination of Reflexes: By Robert Wartenberg, M. D. Chicago, Year Book Pub. Inc. 1945. pp. 222. Price, $2.50.

Material for this book was obtained from an exceedingly thorough, critical survey of the literature, old and new, and included is a bibliography of four hundred and sixty-five reference citations. From all of this one gets a glimpse of the fascinating history of reflexes.

Neuropathologic conditions document themselves readily in reflex changes. The status of the reflexes provides truly objective findings which are the aim of every medical examination.

Heretofore the student of reflexes has been confronted with varied, confusing and conflicting classifications; a chaotic, arbitrary nomenclature; a mystic maze of terms; a staggering number of reflexes.

On the basis of a few simple fundamental physiologic tenets an attempt has been made here to classify and unify the numerous reflexes with the idea of simplifying, clarifying and facilitating the every-day neurologic examination.

Dr. Wartenberg says: ". . . the goal of methods of reinforcement is to make a slight reflex more apparent and one seemingly lost perceptible. It is a common mistake to regard a reflex as lost when it is not . . . The deep reflex is a delicate and sensitive mechanism and is highly susceptible to the slightest external influence . . . the tabetic patient who shows no reflexes when sober may manifest them while under the influence of alcohol, and that the knee jerk, for instance, increases the moment the patient falls asleep and decreases when he is in deep sleep."

The author has achieved completeness, relative brevity, and simplicity of presentation in this excellent, significant and profound scientific work.

C. P. MAY, M. D.

The Marihuana Problem in the City of New York; Sociological, Medical, Psychological and Pharmacological Studies: By the Mayor's Committee

on Marihuana. N. Y. The Jacques Cattell Press, 1944. pp. 220. Price $2.50.

This book is the report of a committee appointed by the mayor of New York upon recommendation of the New York Academy of Medicine to investigate the sociologic and scientific aspects of the use of marihuana. The investigation was undertaken as a result of the extensive widespread publicity on the use of the drug and of its harmful effects. The findings of special investigators of the police department formed the basis for the sociologic report. In New York the distribution and usage occurs principally in Harlem. It was concluded that marihuana was not a determining factor in the commission of major crimes and probably of little etiologic importance in crime in general. Although it was used by school children the incidence of this was much lower than one would be led to believe by popular reports and the smoking of marihuana was not a problem in juvenile delinquency.

The clinical studies were carried out on 77 prisoners in the municipal hospital on Welfare Island. Over half of the subjects had been marihuana smokers. The administration of the crude drug and purified principles in various forms and dosages was carried out under carefully controlled conditions. The mental and physical reactions of the subjects are described. Psychotic episodes occurred in only nine cases and in six instances they were evanescent in nature. In the three cases of prolonged psychosis, in view of the previous and later history, the drug action was considered to be not more than a contributing factor. The acute changes in organic and systemic functions were relatively slight. An increase in pulse rate, blood pressure, blood sugar and metabolic rate were commonly observed and were interpreted as secondary reactions to central nervous system excitation.

Psychologic studies were conducted with respect to effects on psychomotor responses, intellectual functioning, emotional reactions and personality structure. Positive effects were impairment of static equilibrium, hand steadiness and complex hand and foot reactions. Contrary to most descriptions of action, the estimation of time intervals and linear distances were unchanged. Intellectual functioning in general was impaired but the basic personality structure of the individual did not change.

No evidence was found that marihuana produces addiction since it can be stopped without mental or physical distress comparable to that encountered in the morphine addict and little, if any, tolerance is acquired. The effect of the drug on withdrawal symptoms in the morphine addict was investigated in 28 subjects with 28 controls. The impression was gained that the treated subjects were benefitted but the results cannot be considered conclusive. In a comparison between users and non-users of marihuana who served as subjects no evidence was found that the former were inferior in intelligence or had suffered any mental or physical deterioration.

In a chapter on pharmacology by Dr. S. Loewe, previous findings are reviewed and original investigations of the author are described with particular reference to relationship between chemical structure of active principles and biologic activity and to methods of bioassay. Tetrahydrocannabinol is probably the most important active constituent but there is evidence of others which produce certain types of activity.

Considerable time and effort has obviously been expended in this study and pains have been taken to conduct the clinical and psychologic experiments in a scientific manner. Since, however, the subjects were under such controlled conditions the impression gained, of the relative harmlessness of marihuana, may not be justified.

RALPH G. SMITH, M. D.

The Chemistry and Pharmacy of Vegetable Drugs: By Noel L. Allport. Brooklyn. Chemical Publishing Co., Inc., 1944. pp. 252. Price, $4.75.

This is a synopsis of miscellaneous, superficial information more or less chemical and pharmaceutical on selected vegetable drugs, mostly those included in the B. P. Historical notes and therapeutic comments are included.

The chemistry in the case of most of the drugs consists in naming some of the active constituents and stating the chemical or pharmaceutical group to which they belong; e. g., it is stated "Twenty-five alkaloids have been isolated from opium"; of these six are named and the empirical formulae are given for only two. In only a few instances are the structural formulae of the active constituent given. Some of these few are incorrectly written; e. g., the formula given for ephedrine lacks two double bonds in the ring. Chemical tests for identity and assay procedures are given in detail.

The pharmacy consists of description of collection and of manufacturing procedures. A list of the principle preparations of each drug is given with statement of other ingredients and comment as to therapeutic use. A gross description of each crude drug as it appears on the market is included. In most cases the microscopic characters of the crude drug are not mentioned.

The authorities for the botanical means of the plant sources are, without exception, omitted. The plant family is not often given.

The therapeutic comments are perhaps the weakest part of the book. They are in many cases very misleading as: "The physiological action of opium is essentially hypnotic"; "Codeine does not induce drug habit"; "The sulfates of both atropine and hyoscyamine are used for making solutions for hypodermic injection, the former for the re-

lief of pain in sciatica and as an antispasmodic in asthma, the latter for severe nervous excitement"; and "Strychnine itself is so extensively used in medicine both as a tonic and as a vasoconstrictor in cases of cardiac failure . . ."

It is not recommended for those who are not familiar enough with the subject not to be misled and would not add much to the knowledge of those who are that familiar with the subject.

FOSTER N. MARTIN, JR., M. D.

Ventures in Science of a Country Surgeon: By Arthur E. Hertzler, Halstead, Kansas. pp. 304.

Dr. Hertzler is a most interesting, one might almost say fascinating writer. His various monographs have appealed to a large number of medical men both because of their scientific content and their literary style. His "Horse and Buggy Doctor" was a best seller several years ago and had an enormous circulation, hence as an author he is appreciated by the laity as well as by doctor of medicine. His latest work should prove of tremendous interest to physicians in general and more particularly it could be read with enormous profit by the younger medical man. Hertzler has an inquiring mind and has delved into many new things and modified many old procedures, sometimes with success, sometimes without. Even if he failed to succeed in a study or a project he learned something from it and in this book he tells of his successes and of his failures in his ventures in science or more properly into experimental surgery, medicine, and physiology.

One minor criticism of Dr. Hertzler is that he trades in on living in a small town and calls himself a country surgeon. He may be a country surgeon in that he practices in the country but he has traveled extensively, he has studied in Europe, he has taught in one of the larger medical schools in this country and the physical setup of his hospital, his workshop, is as elaborate and complete as any hospital in the United States. In his early years he may have had to do a considerable amount of improvisation, but certainly for the last good many years his institution, although in a smaller town, has every facility and attribute that one would expect in the largest hospitals in the bigger cities in the country.

J. H. MUSSER, M. D.

PUBLICATIONS RECEIVED

C. V. Mosby Company, St. Louis: The Care of the Neurosurgical Patient, by Ernest Sachs, A. B., M. D.

Biodynamica, Normandy, Missouri: Injury and Death of Bacteria by Chemical Agents, by Otto Rahn.

W. B. Saunders Company, Philadelphia and London: Bacillary Dysentery, Colitis and Enteritis, by Joseph Felsen, B. A., M. D. Clinical Biochemistry, by Abraham Cantarow, M. D. and Max Trumper, Ph. D., 3rd ed. Facial Prosthesis, by Arthur H. Bulbulian, M. S., D. D. S., F. A. C. D.